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INTRODUCTION

The articles presented in this volume, contributed by members of the instructional and laboratory staff of the Department of Pathology and Bacteriology (including the Bacteriological Laboratory of the Iowa State Board of Health) are based upon research work. Several represent detailed studies, including a review of the literature on the subjects of interesting pathological conditions which have come to the laboratory. An article representing a statistical study of some of the examinations made in the Bacteriological Laboratory of the State Board of Health is also included. It is hoped to issue successive volumes of contributions as material accumulates. We shall be pleased to be placed on the mailing list of those who issue contributions or transactions dealing with the subjects of Pathology and Bacteriology.

Laboratory of Pathology and Bacteriology.

Iowa City, Iowa, July 18, 1908.

HENRY ALBERT.

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THE STUDY OF A SMALL EPIDEMIC (FOURTEEN CASES)
OF TRICHINOSIS DUE TO THE EATING OF
BOILED HAM. SPECIAL REFERENCE
TO THE OCCURRENCE OF
EOSINOPHILIA.

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HISTORY OF EPIDEMIC:—During the month of October, 1907, an outbreak of trichinosis affecting fourteen persons occurred at Grinnell, Iowa. Thirteen of these were students at Iowa College located in that city. The infection occurred from the eating of boiled ham in one of the clubs and at one of the restaurants. The ham in all cases was obtained from the same packing house and represented meat which had been inspected. Ten of the cases occurred among the students at one boarding club, three were young women students who in company with three others partook of ham sandwiches at a local restaurant, and one was a young man (not a student) who obtained ham at the same place. Most of the cases were diagnosed as trichinosis very soon after the beginning of the symptoms. The occurrence of so many cases among the members of the boarding house; the eating of boiled ham together with the rather characteristic symptoms made the diagnosis comparatively easy. Some of the cases were considered as typhoid, one as rheumatism, and one as a disease of the eyes, until the diagnosis became very evident as determined by an examination of the blood.

The accompanying chart presents in tabulated form the important data of most of the cases:

THE OCCURRENCE OF TRICHINOSIS:—Trichina infection is by no means rare. It occasionally makes its appearance in small epidemics when the diagnosis is as a rule easily made.

The condition has no doubt many times been mistaken for typhoid fever, rheumatism, ptomaine poisoning, or some obscure affection. That isolated cases no doubt frequently go unrecognized is evidenced by the fact, that of 505 unselected autopsies in which the death of the patient was due to some condition other than trichinosis, H. U. Williams of Buffalo found trichina in 27 or 5.3% of the cases. According to statistics collected by C. W. Stiles there were reported up to 1893 a total of 709 cases of trichinosis of which 129 were reported from New York, 119 from Illinois, 115 from Massachusetts, 108 from Iowa, etc. During the past five years a number of cases in this state has come to the notice of one of us (Albert). It has been noticed that most of the cases of trichinosis occur among people, more especially Germans, who are in the habit of eating smoked, salted or pickled meat without having it cooked. A few years ago we received specimens of blood from Dr. Grant Augustine of Minden, Iowa, which on examination revealed the presence of a large number of eosinophile cells, the percentage reaching from 25 to 50. Under date of March 17th, 1906, the following letter referring to eleven cases which had occurred in his practice, was received from Dr. Augustine:—

“In reply to yours of the 15th inst., beg to say that in the last five years I have had eleven cases of trichina infection, all of which came from eating smoked sausage without cooking. In the cases I have had, the first symptoms developed from five to ten days after eating the sausage, and in practically all cases they were of gastro-intestinal irritation. Intestinal pain, pain in the back, diarrhoea, sometimes dizziness, with slight nervous manifestation. In one case constipation was absolute for about one week which was the only exception to the rule. Pulse is generally rapid and small, 120 to 160 or 170; temp. 102 to 106. A stiffness of muscles is observed in 4–6 days after the gastric and intestinal disturbance. A prickling sensation in the muscles is very common. In one case I had a boy 10 years old; it was hard to convince him that his bed did not contain tacks or something sharp to stick him. Swelling of the face is present in nine cases out of ten. No hard nodules appeared in subcutaneous tissue or any place else in the body. The severity of the illness seems to depend upon the amount of infection taken into the stomach. Some cases run a mild course of about 3–4 weeks while others who give a history of having imbibed a little

more freely have very severe symptoms indeed. None of the cases resembled typhoid. Onset is generally sudden. Colicky pain in abdomen, rapid pulse and sudden high temperature.

"Beg to say we are all Germans here and like to eat our pork raw, also are fond of a "rare" Hamburger steak, hence I do a large business during the year capturing the *Tinea solium*. I have examined a great deal of meat containing trichina and experience has taught me that the measle is more plentiful in the neck and about the head, next to this the shoulder or along the back. Have had pork showing trichina in the shoulder when I was unable to find the same in the ham."

The letter is given in full since it presents in an excellent manner the symptomatology of the disease. Since that time Dr. Augustine has reported several additional cases.

DIAGNOSIS OF TRICHINOSIS:—It is not the purpose of this article to refer in detail to the diagnosis and differential diagnosis of trichina infection. Suffice it to say that many cases of trichinosis have gone unrecognized—some of them have been diagnosed as cases of typhoid fever, others as muscular rheumatism and still others as obscure conditions. This appears very evident from the investigation made by Williams cited above. Often during the acute attacks the clinical symptoms are not always sufficiently distinct so as to enable a positive diagnosis to be made. The search for the parasite in the feces does not always give positive results. The examination of a piece of the affected muscle for the embryonic worms is often unsatisfactory—since in the first place the consent cannot always be secured for obtaining a piece of the muscle and in the second place they are by no means always found even in cases where they exist. The occurrence of the eosinophilia therefore has become of more practical value in the diagnosis of this disease. The blood should be examined during the second and third week after infection, for it is then that the eosinophilia is usually at its highest. It is true that eosinophilia may occur in connection with other disease processes. In none, however, does it as a rule attain such heights as in trichinosis, nor does it occur in any conditions which are liable to be confused with infection with this parasite. The conditions in which it has been found especially are as follows:—

- 1 Bronchial asthma in which the percentage of eosino-

philia during and immediately following the paroxysms may be as high as 25%.

2 In many infections of the skin especially pemphigus, psoriasis and other chronic conditions. In these an eosinophilia of 10 to 17% has been noticed.

3 Diseases of the bones are sometimes associated with a mild eosinophilia. Thus the occurrence of an eosinophilia during the course of sarcoma suggest a secondary involvement of osseous tissue.

4 Intestinal parasites:—Eosinophilia has been observed in connection with infection with *Uncinaria*, *Oxyuris*, *Ascaris* and the *Taenias*. The eosinophilia in these conditions may vary from ten to twenty or in rare cases even a higher percentage.

5 *Trichina* infection:—It is in this condition that we find the most constant and altogether most marked eosinophilia. It begins as a rule about the 9th to the 12th day after infection, rising rapidly from the normal 2-4% to 35-50%, and is at its height as a rule about three weeks after infection. The eosinophilia therefore is found to correspond to the time when the embryonic forms of the worm are migrating from the intestine and becoming localized in the muscle. The origin of the eosinophile cells and the cause of the eosinophilia has been a matter of some dispute and will be considered while reviewing the literature on the subject.

Thomas R. Brown,¹¹ of Baltimore, was the first to call attention to the large percentage of eosinophiles found in the blood in trichinosis. His report appeared in April, 1897. A good review of the cases of trichinosis reported in the United States up to July, 1897, is found in the *Journal of the American Medical Association*, 1897, Vol. 29, page 59. It is written by Packard²⁵ of Philadelphia. Thayer³⁷ and Cabot¹² later in 1897 also reported the finding of eosinophile cells in the circulating blood in trichinosis. In 1898 Brown again reported his finding of eosinophilia, describing two cases observed in the medical clinics of Johns Hopkins Hospital at Baltimore. At the time of entrance the percentage of eosinophilia was 37. In one case the percentage reached 68.2. The three conditions to which Brown especially calls attention are the marked eosinophilia, marked

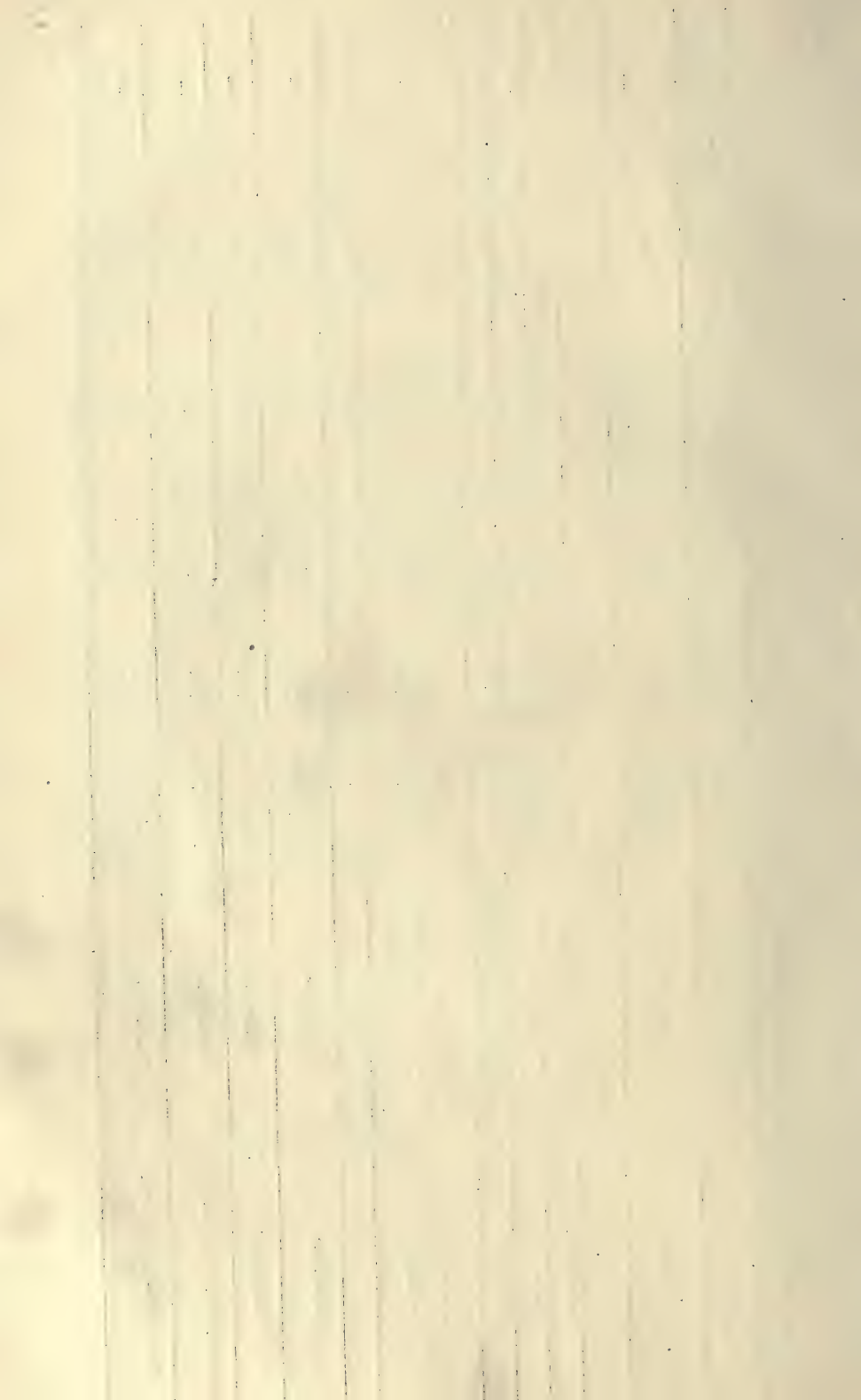
Chart Tabulation of Grinnell Epidemic of Trichinosis

Case	Age	Sex	Source	Date of Infection	Date of Beginning of Illness	Time between Infection and first evident Symptoms	Symptoms (general)	Ultimate recovery, (slow or rapid).	Duration of severe part of illness
1. H. L. B.	20	Male	Boiled ham at club	Oct. 6, 1907	Oct. 18, 1907	12 days	Sore and stiff on rising in morning as with a cold. Muscles of neck, arm, shoulder, calf of leg sore, lame. Terrible aching in small of back. Much constipated.	Rapid	10 days
2. O. C.			"	"	" 10 "	4 "	Diarrhoea, followed a week later with calves sore and lameness. No lasting effects.		
3. H.	21	"	"	"	" 15 "	9 "	Symptoms suggestive of typhoid fever.	Rapid	10 days
4. J. L.	23	"	Ham sdwch. at restaurant	About Oct. 5	" 20 "	15 "	Sick at stomach; constipated, muscles weak.	Slow	
5. Ma.	20	"	Boiled ham at club	Oct. 6	" 15 "	9 "	Swelling of eyes, sore diaphragm. Treated for eye trouble.	Rapid	
6. R. M.	23	"	"	Oct. 6	" 13 "	7 "	Diagnosed as typhoid; fever ran up to 103 lasted 10 days. Treated first for eyes which were swollen. Constipated. Aching in muscles, especially in thighs. Four weeks after first had apparently a second attack.	Slow	10 days
7. Mur.	20	"	"	"	" 16 "	10 "	Soreness in muscles and diaphragm. Constipated.	Rapid	3-4 days
8. R.	20	"	"	"	" 15 "	9 "	Swelling of eyes; soreness in back neck and side. Constipated. In bed with fever 11 days. Temp. 100 1-2 to 102 degrees.	Rapid	14 days
9. S.	21	"	"	"	" 15 "	9 "	Eyes swollen shut. Muscles of calves and shoulders sore and aching; could hardly walk; Constipated.	Slow	14 days
10. T.	20	"	"	"	" 15 "	9 "	Soreness and aching of muscles especially back of neck; eyes swollen. Much constipated. fever 102 degrees.	Rapid	14 days
11. M.	20	female	Ham sdwch. at restaurant	Oct. 4	" 15 "	11 "	"Terrible aching."	Slow	5-6 days

Continued on Page 8 (b)

BLOOD EXAMINATIONS

Symp'toms, Nov. 25, 1907	Clinical Diagnosis	Date	No. Leuco- cytes	Percent eosinophiles	Pe cent polymorphonu- clear neutrophils	Lympho- cytes	Baso- philes	Widal reaction for typhoid fever
None	Trichinosis	Oct. 28, 1907 Nov. 21, 1907 Dec. 18, 1907 Jan. 22, 1908	9,000 8,200 7,300 8,000	72 24 16 6	16 42 62 72	12 32 22 22	2	—
Rather weak	Typhoid (later trichinosis)	Nov. 9, 1907	8,000	24	59	16	1	—
"Feels bum"	Trichinosis	Nov. 5, 1907 Nov. 21, 1907	14,000	4	76	20		—
About well	Eye trouble (one physician Trichinosis (another physician)	Nov. 1, 1907	5,400	22	51	27		—
Weak, muscular work makes muscles sore. Dropped out of college.	Typhoid (one physician) Trichinosis (another physician)	Nov. 5, 1907 Nov. 11, 1907 Nov. 29, 1907	12,000 6,000 6,000	55 40 18	24 36 60	21 24 22		—
	Trichinosis	Nov. 27, 1907	6,900	24	50	24	2	
Almost none	Trichinosis	Nov. 7, 1907 Nov. 26, 1907	9,600 12,000	10 16	70 60	20 24		—
"Rheumatic"	Trichinosis	Nov. 27, 1907	9,200 *	12	52	32	4	—
Soreness of muscles until Nov. 20.	Trichinosis	Nov. 27, 1907	8,600	12	58	30		—
Run down—dropped work	Trichinosis	Oct. 29, 1907 Nov. 5, 1907 Dec. 18, 1907 Jan. 22, 1908	11,000 9,000 8,800 7,800	18 12 6 4	56 64 66 60	26 24 28 32	4	—



leucocytosis and a reduction in the number of neutrophiles. Brown at first believed that in this disease the neutrophiles were transformed into eosinophiles in the muscle. He based this belief principally on the fact that when he first examined the muscle he found transition stages between neutrophiles and eosinophiles, whereas when he examined it two weeks later there was a marked increase of the eosinophile cells. His theory to explain the transition was that the local inflammation caused by the embryos entering the muscle cells caused a marked migration of the leucocytes to such areas. By virtue of their phagocytic activity they ingest bits of degenerated muscle tissue which Brown believed changed the chemical composition of the cell and caused the transformation of neutrophile into eosinophile granule. These cells then wandering back into the blood stream accounts for the marked eosinophilia. According to a more recent (1902) report by Brown, he leans favorably to the well known theory of Ehrlich, according to which the eosinophile cells of the blood are derived from the eosinophile cells of the bone marrow, their appearance in the blood being due to some chemotactic influence. He still however holds to his original belief that some of the eosinophile cells are developed at the site of the lesion.

Atkinson ¹ described a case in 1899 in which a diagnosis of trichinosis was based on the edema together with a marked leucocytosis and an eosinophilia of 50%. In his case the eosinophilia gradually decreased as the symptoms subsided. The diagnosis was verified by the finding of a few non-incapsulated trichina in a section of a muscle.

In June, 1901, Dr. Da Costa ¹⁵ reported a case of a man who developed pain in the calf of the right leg. An operation was performed and a piece of the muscle removed. This on examination was found to contain a large number of trichina. A blood examination was then made with a leucocyte count of 17,600, 3% of which were eosinophiles. The blood examination was made about a year after the pain and it does not therefore indicate whether or not eosinophilia ever existed. The disease was limited to the part mentioned and the diagnosis was not made until microscopical sections were examined.

Schleip ³² gives an interesting description of an epidemic of trichinosis which occurred in Homberg in September, 1903. In all 130 persons were affected. The diagnosis was made very soon

after the appearance of the disease. He made a blood examination of some of his cases, all of which revealed an eosinophilia.

Early in 1904, Opie²⁶ performed experiments by feeding guinea pigs with *Trichina spiralis* and finding a marked increase in the number of eosinophiles in the blood. He did not find any constant alteration in the number of cells until about the end of the second week after infection when their relative and absolute number increased rapidly reaching the maximum at the middle of the third week. This marked increase in the number of eosinophiles corresponds with the time when the embryonic trichinae are being transmitted from the intestine to the muscles by way of the lymph and blood streams. He found an accumulation of eosinophiles in the mesenteric lymph glands. He also noticed characteristic changes in the bone marrow—the fat was gradually replaced with cellular elements and the number of eosinophiles was enormously increased, especially the eosinophilic myelocytes. Opie noticed that pork with 2,000 to 3,000 trichinae failed to produce death of a guinea pig, or did so only after several weeks. A larger dose is usually fatal in from one to two weeks. In all cases he noticed that there was a marked diminution in the number of eosinophiles sometime before death and that in severe infections no eosinophilia whatever appeared. In this connection it may be of interest to notice that Williams and Bentz³⁸ did not find any noteworthy increase of eosinophile cells in the blood of rats and cats artificially infected with trichina; on the other hand Fox, working in the Histological Laboratory of the University of Iowa, noticed not only an eosinophilia of the blood but also the characteristic change noted above in the bone marrow in two cats which had been fed on the refuse of a slaughter house.

Straubli³⁵ reports seven cases, four of which terminated fatally. He notes that the eosinophiles begin to appear about nine days after the ingestion of the infected meat. In the cases which terminated fatally he found Kernig's sign present and knee jerks absent. In all of his cases, the diazo reaction was pronounced—a point of special interest because of the possibility of mistaking trichinosis for typhoid fever. He performed experiments on rabbits. In these he found a large number of the embryos in the heart's blood. His experiments confirmed his previous assumption based on his clinical cases that a rapid destruction of lymphocytes is a bad sign. He regards the eosinophilia as a reaction on the part of the organism to substances given off by the embryos or the degenerated muscle tissue. He

found no local increase of eosinophiles in the muscles invaded by the parasites.

It is of great interest to know that the epidemic which we are reporting was the result of eating boiled ham. It is well known that the majority of cases result from the eating of uncooked seasoned or smoked pork or sausage and we have not been able to find in the literature any instances of cases of trichinosis being traced to the eating of our popular boiled ham.

On making inquiry of several of our largest packing houses in regard to the length of time that their ham was boiled we received from one the following answer:—"When cooking boiled hams we keep the temperature of the water about 190° to 200° Fahrenheit for an hour; then permit the temperature to run down gradually to about 155° to 160° F. when the cooking is finished. Our rule is to cook the hams 38 minutes per lb of weight, that is a 10lb ham would be cooked about 6½ hours." Another wrote as follows:—"We believe that the temperature of 170° F. destroys the effectiveness of any trichinous germs. We always see to it that this temperature and higher is reached in our ham boiling vats. It is to the short sighted financial interest of one who is boiling hams for the market to cook to the minimum amount, in order to avoid shrinkage in weight." From a third we learned the following:—"Our hams are boiled at various temperatures according to size and the demand of the trade in different territories. I should say that the average temperature is between 200 and 212° F.

Just what temperature was used for the boiling of the ham which was the cause of the outbreak which we are reporting it has been impossible to learn, the firm from whom it was obtained stating that their hams are boiled at various temperatures according to the size of the piece and the demand of the trade. They state however that the average temperature is between 200 and 212° F.

"On page 211 of Ainsworth Mitchell's Book on Flesh Food, the statement is made that trichinae perish above a temperature of 69° C. or 140° F." On the other hand the statement is made by Beecher ² that a temperature of 180° F. will not always kill trichina. It is very improbable that boiled ham is at all a common source of trichina infection. If it were, cases of such would be far more frequent. That it may occur, however, is evident from the history of our epidemic. It is probable also that many of the isolated cases of trichinosis especially of the milder types

go unrecognized as such. It appears evident that we are not yet absolutely certain as to just what temperature, maintained for what length of time and varying to what degree with the size of the piece should be attained in order to be absolutely certain that all trichina have been killed. There is little doubt but that it will depend somewhat upon the degree of incapsulation of the embryonic forms and the nature of the capsule, whether it is simply a fibrous one or whether it has become calcareous. This is a subject which would be well worth investigation inasmuch as trichina infection of hogs is of frequent occurrence (it has been estimated that about 2% are infected) and inasmuch as our Government no longer inspects meat for home consumption for the presence of these parasites. Cooking seems to be the only effective prophylactic measure against trichina infection. Thorough salting and hot smoking also generally kills the parasites. They have been found alive in pickled meat for fifteen months, and have been known to resist freezing for two months.

CONCLUSIONS

1 Trichinosis is no doubt of far more frequent occurrence than is usually supposed. Many cases are no doubt diagnosed as cases of typhoid fever, rheumatism, ptomaine poisoning, cholera morbus, etc., or considered as obscure conditions.

2 Trichinosis usually results from the eating of uncooked seasoned or smoked pork or sausage, but may result from the eating of boiled ham which has not been exposed to a sufficient temperature for a sufficient length of time to kill all of the parasites.

3 A temperature of 170° to 200° F., maintained from one to six or more hours depending upon the size of the ham, no doubt destroys the trichinae in the vast majority of cases. Investigations made to determine exactly just what temperature maintained for what length of time is necessary to kill trichina, should be made.

4 The severity or the case varies with the number and no doubt also the vitality of the parasites. In the outbreak which we reported although the symptoms were in many cases quite severe there were no deaths. The usual mortality ranges in different outbreaks from 1 to 30 %.

5 Every means should be adopted to prevent infection of

hogs by removing, if possible, the possibility of their eating dead rats.

6 All pork consumed in any other form than boiled ham should have all parts raised to the boiling point.

7 The diagnosis of trichinosis occurring in isolated cases is frequently a matter of difficulty and often can be greatly assisted by means of an examination of the blood.

8 So far as we know at the present time there is no condition in which the percentage of eosinophiles in the blood reaches such a high number, so constantly as in trichinosis.

9 Eosinophilia occurs in practically every case of trichina infection. A few cases have been reported where there was no eosinophilia (Da Costa, Howard, Rosenburger). Some of these were cases of severe infection and in others an examination was made too long after the time of acute symptoms to be certain whether or not eosinophilia was or was not present at some time.

10 The eosinophilia of trichinosis varies ordinarily from 10% to 60%.

11 The highest percentage known to us was in one of our own cases in which it reached 72%. The next highest of the reported cases is 68.2% (Brown).

12 Eosinophilia makes its appearance with the beginning of the acute muscular symptoms which represents about the 7th to 12th day after infection. It is at its highest at the height of the acute muscular symptoms which is ordinarily during the second or third week after infection. After this time it gradually disappears so that at the end of the second or third month there is usually no increase in the number of these cells.

13 The total leucocyte count is usually, but not invariably increased in trichina infection. The number of neutrophiles is relatively and sometimes absolutely diminished during the period of eosinophilia.

In conclusion, we desire to express our thanks to the physicians of Grinnell, Iowa, who assisted in securing specimens of blood and to E. J. Ringena of Iowa City, to whom we are indebted for assistance in making the differential blood counts.

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ON THE TECHNIQUE OF BLOOD EXAMINATIONS

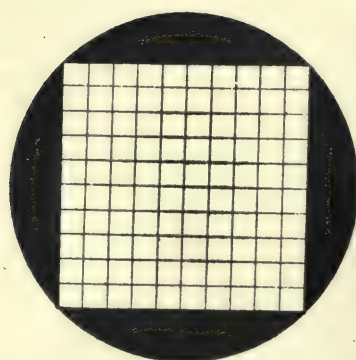
(Observation on the Counting of Leucocytes, Staining of Blood and Marking of Slides, having especially in view greater Ease and Accuracy in the making of Blood Examinations.)

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I COUNTING OF LEUCOCYTES:—The Thoma-Zeiss hemocytometer has become the standard apparatus for determining the number of red and white corpuscles in a given volume of blood. For counting leucocytes, the special white diluting pipette is usually employed, although very satisfactory results may be obtained by using the red pipette and having one and the same dilution serve for the counting of both the red and white cells. When doing so, the diluting solution should contain some aniline dye as methyl-violet for staining the nuclei of the white cells. Such a solution is Toison's. The use of the hemocytometer, although very satisfactory in the hands of one experienced in its use, is, we must admit, still considered by many practitioners as being too complicated and that its use often leads to markedly variable and erroneous results. Regardless of the variable results, the use of the hemocytometer requires considerable time, and when we consider that in order to be of value in surgical practice, the leucocyte count of a given case must be made a number of times, the time element is often deemed of sufficient importance to prevent many who would otherwise take such into consideration from making use of the information furnished by a leucocyte count. Many attempts to gain an idea of the leucocyte count by a direct examination of blood, spread and stained on a slide, have been made. A general survey of the microscopic field will of course give some notion as to the relative proportion of red and white cells but the estimate thus obtainable is too variable to be of value in assisting in the diagnosis or prognosis of inflammatory processes. We have also been told that when examining a spread of blood as ordinarily made, and using a certain eyepiece and a certain objective, that one may expect to find about 500 red cells in a field which means that if on an average more than one white corpuscle is found in a field, it may be taken as evidence of a leucocytosis of

more than 10,000; the degree of leucocytosis could be determined by the number of white cells counted. This method I have used with results about as variable and inaccurate as when making a general survey of the entire field. One of the principal reasons for such unreliable results is the fact that with the usual method of making blood spreads—that of touching the drop of blood with a slide or coverglass and spreading it with the edge of another slide or coverglass held at an angle of about 45° to it, and gently drawing it over the surface of the one on which the spread is to be made, there is neither a uniform spread of the blood nor a uniform distribution of the various cells. The large cells—namely the majority of the white cells do not slip out under the edge of the spreading glass as readily as do the red corpuscles and it will be found that there is a proportionately larger number of leucocytes at the edge and at the end last spread than in the remainder of the field. It is at once evident that a method of determining the leucocyte count by a direct examination of the slide preparation to be of value must consist either of counting all of the cells of the spread or of making a spread which is uniform and the various cells evenly distributed. The former would be a very tedious procedure—the latter may be quite satisfactorily accomplished by making spreads on slides by means of narrow strips of tissue paper such as may be conveniently obtained in the form of cigarette paper. Not all cigarette paper is equally well adapted for this purpose. It must not be too absorbent, nor too oily or stiff. An ordinary piece is cut in two lengthwise. The slide which must be perfectly *clean* is made to touch by its surface near one end, a *small* drop of *freshly* oozing blood; one end of the strip of paper is then laid on it and immediately drawn over the slide while held in a position almost horizontal to its surface. If the drop is too large, the spread will be entirely too thick. One about the size of the head of a match is about right. Experience, however, is a better guide. The spread is then stained, preferably by some method which causes the nuclei of the white cells to be clearly recognizable. For such, I prefer the use of an eosinate of methylene blue dissolved in methyl alcohol such as Wright's or Jenner's stain. Staining with hematoxylin and eosin following fixation with methyl alcohol is also very satisfactory. We now have a spread of blood in which the spread is uniform and the various cells are quite evenly distributed. The counting of the white cells by fields without the actual enumeration of the white cells is liable to give very misleading results. I have therefore had prepared a micrometer eyepiece consisting simply of a circular piece of glass which may be



dropped into the ordinary eyepiece of a microscope. This eyepiece is so ruled that it divides the visible field of the microscope into 100 equal squares as shown by the illustration. We are now ready for making the counts with the object of determining not only the relative number of leucocytes but also the differential leucocyte count. For counting, I prefer to use the $\frac{1}{2}$ objective and preferably a No. 2 eyepiece, although a No. 1 eyepiece may be used with as great satisfaction. The number and kind of leucocytes in a given field may be determined by a mere glance.

The following table illustrates the method of keeping a record of the counts made:—

Red Corpuscles	Leucocytes	Polymorphonuclear neutrophile leucocytes	Lymphocytes	Eosinophiles	Etc.
300	1	1			
700	3	2	1		
1100					
1550	5	3	2		
1920	6	4			
2210	7		3		
2600	9	5		1	
2940	11	7			

Etc., until one hundred leucocytes are counted.

The number of the red cells of the field may be quite accurately determined by counting five of the average squares and multiplying the figure by 20. If the entire spread is very uniform it will be necessary to count the number of red cells in only several of the fields and by comparison be able to make a very good guess of the number of red cells in the other fields. The count of each field is added to the preceding count until 100 leucocytes have been counted. The percentage of the various kinds

of white cells is represented by the number of cells that have been counted and the proportion of white cells to the red cells may be determined by dividing the red cell count by 100. Going on the supposition that the number of red cells in a cubic millimeter of a person's blood is 5,000,000, the number of white cells may be determined by dividing 5,000,000 by the figure which represents the proportion of red cells to each white cell.

It is of course true that we can not be certain without an accurate count that the individual has 5,000,000 red blood corpuscles per cubic millimeter. This method does not therefore give an accurate idea of the number of white cells in anemic individuals. On the other hand, we must recognize that it is not the single leucocyte count that is of so much value, especially in inflammatory processes, but rather the comparison of a number of blood counts made at short intervals. When such is all that is necessary, this method gives us a very accurate idea of any increase or decrease in the number of leucocytes that may be taking place, even better than the use of the hemocytometer because the accurate use of that instrument requires an accurate manipulation without which gross errors are liable to creep in.

As advantages, then, of determining the leucocyte count by a direct examination of the microscopical slide as proposed, over the use of the Thoma-Zeiss hemocytometer it may be urged, First,—that the method is much more simple. The technique necessary for making this count is the same as that which is necessary for making the ordinary differential leucocyte count, and it is possible to avoid the use of the diluting pipette, mixing, spreading of the blood on the special slide and the counting of such as is necessary with the Thoma-Zeiss instrument. Second—It is more accurate in the hands of the majority of practitioners who have not received any special training in the making of blood examinations. When observing the simple precautions of having a clean slide, using freshly drawn blood and the proper kind of paper for making the spread, it is almost impossible to make a mistake as far as technique is concerned. With the use of the hemocytometer, however, errors may creep in by improper dilutions, improper mixing, obtaining the blood in an improper way from the pipette and improper spreading on the counting slide.

I have compared the two methods in a large number of cases and find that as far as comparative results are concerned I am able to obtain them just as well by the direct method as by the use of the hemocytometer. In the hands of students who have

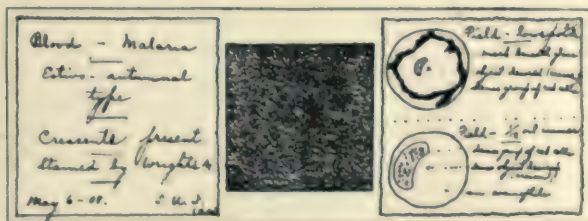
had but little experience in blood examinations, I have found that they obtain much more uniform and accurate results by the method of making the count directly from the stained slide as described. For accurate work or for determination of the absolute number of white cells per cubic centimeter, this method will of course never take the place of the diluting pipette, but for practical work I believe it has distinct advantages.

II. NOTE ON THE STAINING OF BLOOD. Eosinates of methylene blue dissolved in methyl alcohol such as Wright's and Jenner's stain have now become standard blood stains and by many are the only stains used for blood work. We have, however, heard much about variable results and it is such that has lead to the placing on the market of the many modifications which we see at the present time. It has become quite generally recognized that in order to obtain proper results pure methyl alcohol must be used for dissolving the stain. I have been able to obtain the best results when using Kahlbaum's methyl alcohol free from acetone. Another difficulty in the technique has been the precipitate which is frequently found in a stained preparation. This occurs especially when the stain is diluted or washed off with ordinary tap water and may be largely avoided by using distilled water. Even under such conditions the precipitate will sometimes form. It may be removed, however, as has been suggested by different workers by a momentary washing with alcohol (ethyl). I have found, however, that because of the decolorizing effect of ethyl alcohol, the use of methyl alcohol is far more satisfactory. Finally it has been observed that when blood spreads are from several weeks to several years old, they are stained very unsatisfactorily by the use of Wright's stain or any of the similar modifications, due to the fact that the basic stain is especially prominent whereas the acid or eosin stain takes but little effect resulting in a preparation in which the red cells are either practically unstained or are stained a light greenish blue. I have found that by placing several drops of methyl alcohol on the slide thus stained and allowing it to act for 10 to 30 seconds, the eosin stain becomes quite prominent resulting in well stained preparation. The use of ethyl alcohol does not bring out the eosin stain and has a marked decolorizing effect on the basic stain.

III. MARKING OF SLIDES. It frequently happens that physicians and more especially teachers have occasion to demonstrate certain object in slide preparations. These may be of small size, few in number and not easily recognizable, as for

instance, the crescents found in the blood of the estivo-autumnal type of malaria or some particular or peculiar manifestation of a cell. I have seen physicians look over their old slide preparations for hours and not be able to find the object desired. By using the mechanical stage and marking on the slides the place at which the stage should be set facilitates the finding of objects to a certain degree. The mechanical stage, however, is not always at hand, and even with its use a slight change in position frequently makes the searching for the object very annoying. Somewhat more serviceable is the Maltwood finder used with or without¹ the moveable stage.

I have for sometime made use of a simple method which has served me admirably for the finding of objects in microscopical slides. In the first place I make an examination of the field with or without the use of the mechanical stage. If a mechanical stage is not used, the slide should be firmly held in whatever position desired by means of clamps. Whenever I find an object which I desire to be able to find again and the searching for which



may require considerable time, I lower the sub-stage of the microscope and with pen and ink make a dot on the lower side of the slide corresponding to the part opposite the lens above. After the ink has dried I remove the slide from the microscope and by means of a diamond or other hard instrument make a little scratch on the glass around the ink mark. I then wipe away the ink mark and render the scratch on the glass more easily visible by scratching it with a lead pencil. I now have on the under side of the slide a small black ring, within which and about the center of which must be the object the location of which I desire to mark. I then again place the slide on the microscope and with the low power find the black ring. The slide is held by the clamps or mechanical stage in such a position that the center of the ring corresponds to about the center of the field of the micro-

¹ Pepper, W.—J. A. M. A., June 20, 1908, p. 2062.

scope; then by use of the higher powers I again find the object. If the object is not exactly in the center of the ring it may be necessary to move the slide about a little before the object is found. When found, however, it is placed in the center of the microscopic field. I then make a small representation of the field under the high power on a label illustrating in a general way the relation of the particular object to be found to other distinctive objects in the field. I then return to the use of the low power and on the same label make a sketch of the field, marking certain distinctive features as well as the outlines of the ring on the under side of the slide, found by slightly altering the focus. By the aid of such sketches of the microscopical field, all that is frequently necessary is to find the corresponding field with the low power. If the stained preparation has been fixed on a cover-glass and mounted on a slide, it is best to wait until the balsam has become hard, otherwise a little moving of the cover-glass will disarrange the relative position of the object as regards the marking on the under side of the slide.

By means of this simple procedure I have been able to find an object in a half minute whereas without it, it has frequently required a half-an-hour's search. I feel sure that its employment will be found a great convenience to others.

PATHOLOGICAL CONDITIONS OF THE LIVER FOLLOW-
ING CHOLECYSTECTOMY—AN EXPER-
IMENTAL STUDY*

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There is a growing tendency on the part of surgeons to perform cholecystectomy for gall bladder disease when cholecystotomy might be done. This together with the fact that there are no definite conclusions as to the function of the gall bladder, other than that of being a reservoir or store-house in which the bile is kept during the intervals between digestion periods, to be emptied into the digestive tract when needed for carrying out its special digestive function, led me to undertake a series of experiments. Considering the small size of the human gall bladder under normal conditions and the amount of bile and gall bladder fluid excreted and secreted in twenty-four hours, it seems illogical to think that its function is to serve as a store-house for bile and for no other important purpose. Therefore it seemed worthy of trial to perform some experiments along this line, with the view of determining, if possible, the hepatic changes, if any, following cholecystectomy.

The human gall bladder in a state of moderate distention, holds from fifty to sixty c. c. of bile. The average daily flow of bile is twenty-seven and one-half ounces and of gall bladder fluid two and fifty-three-hundredths ounces, as determined by Mayo Robson in extended observations upon a case of biliary fistula where all the bile flowed through the fistula and in a case of gall bladder fistula, not biliary, where only the gall bladder fluid flowed through the fistula. Quoting the above-named author: "It is scarcely consistent with the economy of nature to provide beings with purposeless organs, and since the reputed function of the gall bladder as a reservoir of bile

*Abstract (giving the pathological considerations) of a thesis presented for the degree of Master of Science.

between the periods of digestion is both rational and probable, we may accept the theory until some better one is discovered."

As previously stated, considering that twenty-seven and one-half ounces of bile are excreted daily and two and one-half ounces of gall bladder fluid are secreted daily and since the gall bladder does not hold but one and one-half to two ounces of bile, it does not seem that the function, primarily, could be that of a store-house for bile. Space will not permit detailing the valuable observations made by Mayo Robson on the flow of bile and of gall bladder fluid. He was fortunate enough to be able to carry out his observations on human subjects over a period of fifteen months in one case and eighteen months in another. During this time accurate observations were made hourly, not only as to the flow of bile but also as to the diet used and the general physiological results obtained. A few of his conclusions will be of interest and valuable from a scientific and practical standpoint:

- 1 The bile is probably chiefly excrementitious and like the urine is being constantly formed and cast off.

- 2 The antiseptic properties of bile are unimportant.

- 3 Whatever little antiseptic quality bile may have is probably derived from its admixture with gall bladder fluid.

- 4 The excretion of bile seems to go on constantly and with great regularity.

- 5 The excretion is apparently not materially influenced by diet.

In the case where the bile was all discharged through the fistula externally, no definite digestive changes or disturbances were noted nor did the flow of bile seem to be influenced by diet. Reasoning from this observer's own assumption which at first was that we must accept the theory that the gall bladder's function was to store bile during the intervals between digestion and his later conclusion that the "flow of bile is not influenced by diet," it must be assumed that the one and one-half ounces stored up constitutes the extra demand of digestion and also that the gall bladder has some means other than that exercised continuously of discharging such.

During the course of my experiments, I operated on eight dogs, two of which died soon after the operations. No account of these two will be given. As anesthetics, chloroform and ether, were used—chloroform first, in order to shorten

the period of getting them anaesthetized, then changing to ether to minimize the danger of their succumbing to the anaesthetic. The operations were performed under fairly aseptic conditions; the field of operations being first shaved, then washed with green soap and water, followed by a solution of bichloride of mercury. An incision was made along the right semilunar line down to the peritoneum. This cavity was next opened. I then removed as a "control" a V-shaped piece of liver, about one-half inch wide at the free edge of the liver and about three-fourths inch deep from a lobe distant from the gall bladder. This wedge-shaped piece was placed into a fixing fluid, and was used for histological examination. The V-shaped opening thus made was closed by a single cat-gut suture, the suture entering into the free edge of the liver, about one-third inch from the cut surface, passing deeply into the liver substance, across the apex of the V-shaped opening, and out of the liver on the opposite side at a point corresponding to that of entrance. By tying the suture over the free edge of the liver, the cut surfaces were easily coaptated—no other suture being necessary. Next, cholecystectomy was performed in the following manner:—The fundus of the gall bladder was grasped with forceps, the peritoneal attachment of the liver about the fundus was carefully loosened, and the gall bladder was separated from the liver by a gentle manipulation with the fingers. In this way, tearing of the liver substance to any great degree was obviated. A gauze sponge was packed temporarily over the bleeding surface of the liver to control the slight hemorrhage. Without opening the gall bladder, a silk suture was placed around the cystic duct, and this tied off together with the artery to the gall bladder. The ducts were tied at the distal end, at the point of junction with the gall bladder in all cases except in dog No. VI, in which case it was tied off about one-fourth inch from its junction with the hepatic duct. The gall bladder was excised. No further treatment was applied to the stump. Previous to excising the gall-bladder, forceps were applied to its neck to prevent the bile from escaping into the abdominal cavity. The gauze packing was removed and the wound closed and sutured, without drainage. Chromosized cat-gut continuous sutures were used separately for the peritoneum, transversalis fascia, and muscles, and for the skin silk-worm-gut was used.

No attempt was made to obtain dogs of the same size or age—neither of the same sex. As we find gall bladder disease occurring in people of various ages and under various condi-

tions, it seemed proper to use animals of different age and size and sex. A brief description of the six dogs by number will give a better idea of the animals used:—

Dog No. I, Female; one year old; weight fifteen pounds; white.

Dog No. II, Male; one and one-half years old; weight thirty-five pounds; black.

Dog No. III, Male; eight months old; weight fifty pounds; black.

Dog No. IV, Female; sixteen months old; weight forty-five pounds; brown.

Dog No. V, Male; four months old; weight twenty pounds; black.

Dog No. VI, Male; age two years; weight thirty pounds; black.

In each of the above cases the wound healed nicely and without extensive peritonitis as evinced at autopsy. Their diet was mixed, consisting of bread and a good allowance of meat, with general scraps from the table. Water constituted their drink. The number of days, each animal was kept after operation, is given in the subsequent table. Each dog gained in weight but no accurate record of weights was kept. No attempt was made to examine the urine or fæces, but appeared, on gross inspection, to be of normal color, amount and consistency.

Three investigators—Nasse, Oddi, and De Voogt—have previously removed gall bladders to determine resulting changes. The most extensive work recorded was that done by De Voogt, to which more reference will be made in the following pages. Results obtained by these three investigators seem to have differed. Oddi found a new reservoir for the bile. Nasse found nothing—no new gall bladder, neither did he arrive at any conclusions; De Voogt performed his experiments with a view of determining whether or not nature does not demand a reservoir for the bile and if nature does demand one he maintains that in gall bladder disease, if possible, it is better to do cholecystotomy than cholecystectomy, because, he assumes, that in the new reservoir, stones would be formed and conditions not bettered and since the mortality is greater in cholecystectomy, then cholecystotomy should be the operation of choice. Oddi found a new gall bladder which he considered to be a compensatory distension of the cystic

duct. He also states that the common bile duct was dilated. He used only three dogs, and the period of observation was short, being only one month. Nasse found neither widening of the ducts nor any new reservoir for the gall. He does not state how many animals he operated upon, how old they were, or how long he let them live. Therefore, his experiments are of little value from a scientific standpoint except for the mere statement made by him that there was no dilatation of the common bile duct, nor was a new bile reservoir found. De Voogt used five dogs, one of which died three days after operation. He states that in the four dogs which lived longer, he found in each a new receptacle for the gall, and that the wall of the new gall bladder was much thicker than the original. The bladder was shaped like a pear and tapered toward the cystic duct. The depth of these gall bladders varied from 1.5 c. m. to 3 c. m. and the width of the largest was 1.5 c. m. He states that the silk threads used as a ligature to tie off the cystic duct were found at the bottom of the newly formed gall bladder, partly fixed on the inside wall of the same. These gall bladders were surrounded by liver substance and visible only after having been dissected out. Histologically he found no change in the liver and macroscopically only a depression for the gall bladder and at this point the color was a little more pale. Summing up his work, De Voogt's conclusions are as follows:—

I. That the newly formed gall bladder is derived anatomically from the cystic duct.

II. That it is not due to the reconstructive power of the duct, but rather that the cystic duct is slowly stretched. He maintains that two forces—the inspiratory force and the secretory force cause the bile to flow; that these two are constant, and since the gall does not flow constantly into the intestines but more after eating, the constant pressure behind causes dilatation of the cystic duct.

The four dogs operated upon by DeVoogt lived the following periods:—Dog No. II, lived fifty days; No. III, ninety-eight days; No. IV, fifty-five days; and No. V one hundred and seventy-five days. Dog No. I died the third day and hence is not considered in the study.

Following is a description of the conditions found in my own dogs. To determine these the dogs were chloroformed, operated upon and pieces of liver substance removed while the

dogs were under the same respiratory and circulatory conditions as when the "controls" were obtained. The dogs were then given enough chloroform to kill them and further observations made. Dog No. I was operated upon Dec. 3rd, 1905, and autopsy the following March 18th, after a period of one hundred and five days. On opening the abdomen macroscopic changes were plainly visible. The entire liver had changed its shape and was constricted and nodular, as seen in mild cases of Laennec's cirrhosis. Over the entire liver was a fibrous envelope, a thickened perihepatic capsule. See Fig. I, (a) which is from a photograph of this liver. It shows the nodular appearance of the liver, as well as the perihepatic fibrosis. This fibrosis was found over the entire surface of the liver and was not due to local or general peritonitis since at no place were there any adhesions between the liver and the parietal peritoneum. The cystic duct, left at the time of operation, was one and one-fourth inches long, was partially embedded in liver substance and only slightly dilated. The lumen was still present, the mucus membrane was bile stained, and the opening into the hepatic duct was still patent. Microscopically it was found that the capsule was five or six times thicker than normal (compared with control,) also that this fibrosis dipped down at intervals into the liver substance. The intralobular capillaries were found to be greatly dilated and gorged with blood. These capillaries were in places, as wide as a row of liver cells. The parenchyma cells were on this account, atypically arranged, being compressed by the engorged capillaries. The nucleus of the hepatic cells was often found near the edge of the cell instead of being centrally located as in the control. The endothelial lining of the capillaries was distinct. The main changes found were:—

- 1 Perihepatic fibrosis.
- 2 Dilation of intralobular blood capillaries.
- 3 Atypical arrangement of liver cells.
- 4 Compression and beginning atrophy of a part of the parenchyma cells.
- 5 Cystic duct slightly dilated.

Dog No. II, was operated upon Dec. 17th, 1905; autopsy held the following March 18th, an interval of ninety-one days. Control was normal. On gross inspection no perihepatitis was found, but the cystic duct was markedly changed. There was a new bile reservoir measuring one inch in length and one-half inch in diameter, tapering toward the proximal end. This was

PLATE I



Fig. I.—a and b, livers with perihepatic fibrosis.



Fig. II.—a, newly-formed bile reservoir; b, liver; c, common bile duct; d, duodenum; e, pancreas.

filled with bile and apparently performed the function of a gall bladder. This new gall bladder is shown in figure II (a). Microscopically the capsule was found to be slightly thickened. The intralobular capillaries were dilated and engorged with blood the same as in dog No. I. The main changes in this dog were:—

- 1 Presence of new bile reservoir.
- 2 Dilation of intralobular blood capillaries.
- 3 A typical arrangement of liver cells.
- 4 Compression and beginning atrophy of parenchyma cells.
- 5 Slight perihepatic fibrosis.

Dog No. III, operation Dec. 10th, 1905; autopsy following March 18th, a period of ninety-eight days. Control normal. Macroscopically there was no perihepatic fibrosis. The cystic duct was markedly changed, the new bile reservoir was one and one-eighth inches long, and five-eighths inch in diameter, and filled with bile. This reservoir was almost completely imbedded in liver substance, and resembled a gall bladder. Microscopally the blood vessels were markedly dilated, especially the intralobular capillaries. The endothelial lining of capillaries was prominent. This distention of the capillaries caused atypical arrangement of hepatic cells and also compressed them. (Fig. III, a) represents a photograph of the new gall reservoir and liver of the above described autopsy. The changes found were:—

- 1 Presence of a new gall reservoir.
- 2 Dilation of intralobular blood capillaries.
- 3 Atypical arrangement of hepatic cells.
- 4 Compression and beginning atrophy of parenchyma cells.

Dog No. IV, operation Nov. 30th; autopsy following March 18th, a period of one hundred and eight days. Control normal. Autopsy; macroscopically, there was a perihepatic fibrosis especially marked about the region of the gall bladder. (Fig. I, b) A new reservoir one inch long and one-half inch in diameter was found (see Fig. IV, a). The gall reservoir was imbedded in a dense tissue, (Fig. IV, b) whitish in color on the surface, which extended about an inch on either side of the surface of the gall bladder, gradually shading into liver substance. Three tumor-like masses (Fig. IV, c) were found in the lesser omentum, and upon microscopic examination were found to consist of the same histological elements (sarcomatous) as the tissue surrounding

the newly formed gall reservoir. This case presented the following changes:—

- 1 Perihepatic fibrosis.
- 2 Dilation of intralobular blood capillaries.
- 3 A typical arrangement of liver cells.
- 4 Compression and beginning atrophy of parenchyma cells.
- 5 Presence of new gall reservoir.
- 6 A new growth which arose from the site of operation, apparently traumatic in origin, secondary to the operation. This new growth was a sarcoma of a mixed cell type, which had already given rise to metastatic formations in the lesser omentum.

Dog No. V, operated upon Nov. 7th, autopsy the following March 18th, a period of one hundred and eleven days. Control normal. Autopsy; the stump of the cystic duct, about one-half inch long, slightly dilated, was buried in liver substance. The changes in this case were:—

- 1 Dilation of intralobular blood capillaries.
- 2 A typical arrangement of liver cells.
- 3 Compression and beginning atrophy of liver cells.

Dog No. VI, operated upon Nov. 7th; Autopsy following March 15th, a period of one hundred and thirty-eight days. Control normal. At time of operation, the greater portion of the cystic duct was removed leaving a stump about one-third inch long. At autopsy this stump was found to be dilated into a reservoir about one-third inch in diameter, was filled with bile and had a mucosa like that of a normal gall bladder. The capsule of the liver was only slightly thickened but vascular changes were present the same as in the other five dogs. The main changes in this liver were:—

- 1 Presence of new gall reservoir.
- 2 Vascular changes and resulting parenchyma changes as in all other dogs operated upon and previously described.

Summing up the changes and evidence found in the series of experiments, we find that:—

1 In two of the dogs (Nos. I and IV) we had a thickened capsule of the liver which was not due to chronic peritonitis, inasmuch as there were no adhesions to the parietes, and also since



Fig. III.—a, newly-formed bile reservoir; b, liver; c, cystic duct; d, common bile duct; e, duodenum.



Fig. IV.—a, newly-formed bile reservoir; b, tumor(sarcoma); c, metastases of b; d, cystic duct; e, common bile duct; f, duodenum; g, liver.

the striations of the capsule marked the liver substance off into irregular areas (see Fig. I, b). The contraction of this thickened capsular fibrosis caused a roughened nodular appearance of the liver, and since it is not due to peritonitis, and resembles in appearance a cirrhotic liver (moderately "hob-nailed" in type) the condition might be termed "perihepatic cirrhosis" the exact cause and nature of which must be determined by future experiments along this line.

2 In four of the six dogs there resulted a new reservoir for the bile. Anatomically, this developed from the stump of the cystic duct. It formed regardless of the length of the cystic duct, and without doubt, it was compensatory in the sense of being due to the pressure of the bile behind, regenerative in the sense that following dilatation, the walls of the new gall bladder hypertrophied, following the general laws of dilatation and hypertrophy as exemplified elsewhere in the body. This new gall reservoir is of the same structure as a gall bladder, not necessarily a regeneration however, because we find little difference between the histological structure of a normal gall bladder and that of the cystic duct. The only histological difference if any, is a slight difference in the height of the cylindrical epithelial cells, a matter of very little practical significance in making a differentiation. The question now arises as to what would cause this dilatation and hypertrophy of the cystic stump and whether or not nature demands a gall bladder in those animals where it is normally present. We agree with Mayo Robson that, "It is scarcely consistent with the economy of nature to provide beings with purposeless organs," but do not believe that the primary function of the gall bladder is to act as a reservoir for bile between the periods of digestion. Robson determined that thirty ounces of bile and gall bladder fluid were excreted in twenty-four hours. It seems therefore that the one and one-half ounces of bile, the gall bladder's normal capacity, would be of little importance in storing up bile during the period between meals.

The dilatation was evidently due to the pressure of the bile, the pressure being brought about by:—

- (a) Secretory pressure, which is but slight.
- (b) Inspiratory force, compressing the liver with each inspiration.

3 In dog No. IV, there developed a definite tumor process, a mixed cell sarcoma, evidently traumatic in origin, following

the removal of the gall bladder. The primary site was at the seat of operation, the fissure of the gall bladder, thence it extended in all directions into the liver substance, and secondarily through lymphatic radicles, produced metastases in the lesser omentum.

4 The intrahepatic changes found were also important. DeVogt found at an early period, a dilatation of the biliary radicles. In all of my dogs, (six) killed at a later period, the following changes were present:—

a A dilated condition of the intralobular blood capillaries often dilated to a diameter as great as that of a normal liver cell. The endothelial lining of the capillaries was distinct.

b The pressure in the dilated vessels caused the parenchyma liver cells to be compressed, the nuclei often lying near the free edge of the cell.

c The pressure in the capillaries caused the normal arrangement of the rows of liver cells to be somewhat altered. The biliary radicles were normal.

It seems therefore, that the gall bladder has a function other than that of merely serving as a reservoir for bile between digestion periods. "By its removal we get a biliary stasis" (de Voegt). This retained bile acts as a poison to the hepatic cells, and nature in attempting to free the liver of this poison, produces a hyperemia. The dilated blood capillaries compresses the parenchyma cells, disarranging them and causing pressure atrophy of the cells. Nature, furthermore, attempts to correct the condition by partial regeneration of a reservoir for the bile, but complete restitution of a gall bladder does not take place. The vascularity persisting, it is reasonable to assume that cirrhosis of the liver would result if the animals were permitted to live for a longer time, indeed there was a beginning cirrhosis (Fig. I, a and b) in two of our cases. The following table presents a summary of the experiments:—

Dog No.	Sex	Condition of Control	No. of days between Cholecystectomy and Autopsy	Perihepatic Fibrosis	Dilatation of Intralobular Blood Capillaries	Altered arrangement of Liver Parenchyma due to Pressure	New Bile Reservoir
I	F	Normal	105	Present	Present	Present	Absent
II	M	Normal	91	Absent	Present	Present	Present
III	M	Normal	98	Absent	Present	Present	Present
IV	F	Normal	108	Present	Present	Present	Present
V	M	Normal	141	Absent	Present	Present	Absent
VI	M	Normal	138	Absent	Present	Present	Present

In conclusion, I desire to thank Professors Jepson and Albert for many valuable suggestions while conducting this work.

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THE PANCREATIC REACTION (CAMMIDGE TEST) OF THE URINE

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In March, 1904, P. J. Cammidge of London, England, described a method of examination of urine in suspected cases of pancreatic disease which was considered of great assistance in diagnosis. In May, 1906, he published an improved method which simplified the examination and eliminated some disturbing factors.

I have examined over one hundred fifty specimens of urine by these methods and have obtained a positive reaction from twenty specimens. Seventy-five specimens taken from normal people and from patients suffering from tuberculosis, pneumonia, ulcer of the stomach and several other diseases where the pancreas was not diseased failed to give the reaction.

I will describe these methods as both were used in this series of examinations.

The original method was as follows: Acidify fresh urine (preferably from 24 hours' amount) with acetic acid, boil and filter to remove albumen. Remove sugar by fermentation, boil to remove alcohol and filter. For complete differentiation, two reactions were necessary.

Reaction A. Mix 10 c. c. of urine and 1 c. c. of HCl (c. p.). Boil in small flask for 10 minutes. Add 5c. c. of urine and 5c. c. of distilled water and cool rapidly. Add 4 gms. of lead carbonate and allow to stand till reaction is complete. Filter and wash filter with 5 c. c. of water. Add 2 gms. of sodium acetate and .75 gm. of phenylhydrazin hydrochlorate; boil gently for 4 or 5 minutes; pour into tube and allow to stand undisturbed for 12 to 24 hours. In acute pancreatitis no crystals form by Reaction A—on the other hand, crystals of Reaction A may occur in other conditions than diseases of the pancreas, which is not the case

with Reaction B, which is as follows: Take 20 c. c. of urine and 10 c. c. of HgCl_2 and allow to stand for a few minutes. Filter. Take 10 c. c. of the filtrate and 1 c. c. of HCl and boil for 10 minutes. Add 10 c. c. of water and 5 c. c. of filtrate and cool rapidly. Add 2 gms. of lead carbonate and proceed as in Reaction A. In order to eliminate the possibility of sugar a control is run as in Reaction A except for the HCl step. If crystals are found in the control further steps must be taken to remove sugar.

The crystals obtained by this method should form in 24 hours and examined under a $\frac{1}{8}$ lens are pale yellow and very fine. They may be arranged in fans, sheaves, or in compact masses resembling sea urchins. When a 33% solution of sulphuric acid is run under the cover glass the crystals turn brown as soon as the acid reaches them and gradually dissolve, the time of solubility ranging from a few seconds to 4 or 5 minutes. Those from acute cases dissolve in from 10 to 30 seconds, while those from chronic and malignant cases resist the acid for from 3 to 5 minutes. If undissolved in 5 minutes the crystals are probably not pancreatic.

The following is a list of the cases (with outline of history and clinical findings) giving a positive reaction:—

Case 1. Mr. F. 6-16-05. No sugar or albumen in urine. Reaction A.—Many crystals soluble in 2 to 3 minutes; a few insoluble. Reaction B:—Same as in A. Control—No crystals. Laboratory diagnosis—Chronic pancreatitis. Clinical history. Marked loss of weight, pain and tenderness in epigastrium for several years. Intense jaundice some months before examination and occasional putty colored stools. Frequent attacks of very severe pain. Clinical diagnosis—gall stones. Cholecystotomy was performed to remove stones and drainage established. Little opportunity was offered during the operation for a thorough examination of the pancreas but it was very firm. A large stone weighing $19\frac{1}{2}$ grains was removed from the ampulla of Vater. This stone was blocking the common and pancreatic ducts and had on one side a white incrustation probably from the pancreatic juice. The patient has gained about 40 lbs. and is now enjoying good health. A positive reaction persisted in the urine of this patient since the operation as indicated by three subsequent examinations as follows:—

Second examination: 6-30-05. Reaction A.—Many crystals soluble in 15 to 60 seconds. B.—Same as in A. Control—

No crystals. Third examination 7-20-05. Same result as in second examination. Fourth examination 7-30-05. Reaction A—Crystals soluble in 15 to 30 seconds. B.—Crystals soluble in 1 to 3 minutes. Control.—No crystals.

Case 2. Dr. F. 8-2-05. No sugar or albumen in the urine. Reaction A—Crystals soluble in 2 minutes. B—crystals soluble in $1\frac{1}{2}$ to 2 minutes. Control—no crystals. Laboratory diagnosis—chronic pancreatitis. Clinical history:—Slight jaundice followed by severe hiccoughs lasting 24 hours. Tenderness over lower right chest but no abdominal tenderness. Tympanic note extending as high as the 5th right mammillary line. Liver dullness normal in right axillary line and in back. Temperature gradually rose to $102\frac{1}{2}^{\circ}$ one week after onset of disease when a diagnosis of infection of the gall bladder was made. Operation revealed a ruptured abscess of gall bladder, dense adhesions from liver to colon and stomach causing the pus to migrate upward between liver and diaphragm. One large stone was found free in the abscess cavity. The liver between the mammillary and median lines was thin and was pushed up and back on the diaphragm. Drainage was established followed by an uneventful recovery. Patient is now in good health.

Case 3. Mr. M. 8-11-05. No albumen. Sugar removed by fermentation. A—crystals soluble in 45 seconds. B—crystals soluble in $1\frac{1}{2}$ minutes. Control—no crystals. Laboratory diagnosis—chronic pancreatitis. Clinical diagnosis—diabetes mellitus. No further record of case is obtainable.

Case 4. Mrs. A. 11-5-05. No sugar nor albumen. A—crystals soluble in 45 seconds. B—crystals soluble in 1 minute. Control—no crystals. Laboratory diagnosis—chronic pancreatitis. Patient was sent in with a diagnosis of perforated ulcer of the stomach. Clinical history:—Pain and tenderness in epigastrium, putty colored stools, loss of weight, constipation, and pain in stomach after meals. Operation revealed a gangrenous gall bladder with a stone free in the abscess cavity. Firm adhesions about pylorus, duodenum and head of pancreas. The pancreas was very firm. Uneventful recovery and subsequent good health.

Case 5. Mr. H. 12-22-05. No sugar nor albumen. A—crystals soluble in $3\frac{1}{2}$ to $4\frac{1}{2}$ minutes. B—crystals soluble in $2\frac{1}{2}$ to 4 minutes. Control—no crystals. Laboratory diagnosis—pancreatitis or more probably, malignant tumor. At operation the pancreas was found to be nodular, enlarged and very hard.

Post mortem and microscopical examination showed it to be carcinoma of the pancreas.

Case 6. Mrs. S. 2-3-06. No albumen. Sugar removed by fermentation. A—crystals soluble in 30 to 40 seconds. B—same as in A. Control—no crystals. Laboratory diagnosis—Clinical pancreatitis. Diagnosis—diabetes mellitus. Patient now in good health but still has sugar in urine occasionally.

Case 7. Mr. R. 1-26-06. No sugar nor albumen. A—crystals soluble in 30 seconds. B—crystals soluble in 45 seconds. Control—no crystals. Laboratory diagnosis—pancreatitis. Clinical history:—Profuse gastric hemorrhage. Clinical diagnosis—Cirrhosis of the liver. Patient died in two or three days. No post mortem examination.

Case 8. Mr. C. 6-14-06. No sugar nor albumen. A—crystals soluble in 3 to 4½ minutes. B—no crystals. Control—no crystals. Laboratory diagnosis—chronic pancreatitis. Clinical history:—attacks of gall stones for seven years. Chills with sudden rise of temperature. Many small stones were removed at operation. Later the patient lost appetite for meat and when meat was eaten it passed unchanged. Patient gradually lost strength and died. No post mortem was held. Clinical diagnosis:—Pancreatitis and abscess of liver.

Case 9. Mrs. S. 8-3-06. No sugar nor albumen. A—crystals soluble in 1½ to 2 minutes. B—crystals soluble in 2 to 2½ minutes. Control—no crystals. Laboratory diagnosis—pancreatitis. Clinical history:—Acute attacks of cholecystitis, marked tenderness over gall bladder, occasional vomiting, no jaundice. Clinical diagnosis:—Cholecystitis probably with gall stones. Patient still has attacks of cholecystitis with constant pain over gall bladder.

Case 10. Mr. F. 8-15-06. No sugar nor albumen. A—crystals soluble in 1 to 1½ minutes. B—no crystals. Control—no crystals. Laboratory diagnosis:—Pancreatitis. Urine sent in without data except that it was from a case of suspected gall stones.

Case 11. 12-6-06. Mr. B. No sugar nor albumen. A—crystals soluble in 15 seconds. B—crystals soluble in 20 seconds. Control—no crystals. Laboratory diagnosis—pancreatitis. Urine sent in without data.

Since examining the above cases I have been using the im-

proved method² as suggested by Cammidge which consists of but one reaction and is as follows:—

Remove sugar and albumen as before. Take 20 c. c. of urine (filtered) and 1 c. c. of strong hydrochloric acid (s. p. 1.16) and boil gently on sand bath for 10 minutes. Cool rapidly. Make up to 20 c. c. with cold distilled water. Add slowly 4 gms. of lead carbonate. Allow to stand a few minutes to allow the reaction to be completed. Cool. Filter until clear. Add 4 gms. of tri-basic lead acetate to filtrate, shake and filter until clear. Add 2 gms. of finely powdered sodium sulphate to filtrate, heat to boiling point, cool and filter. Add 8 c. c. of water to 10 c. c. of filtrate and add 2 gms. of powdered sodium acetate and .8 gm. of phenylhydrazin hydrochlorate and 1 c. c. of 50% acetic acid. Boil gently (preferably on a sand bath) for 10 minutes. Filter while hot. Make up to 15 c. c. with hot distilled water. Allow to stand for 12 to 24 hours undisturbed. A control is run same as above except for the "hydrochloric acid" step.

The crystals obtained by this method and which indicate "disease of the pancreas" are similar to those in the first series. When a 33% solution of sulphuric acid is run under cover glass the crystals turn brown as soon as the acid reaches them and rapidly dissolve in 10 to 15 seconds.

Case 1. Mrs. B. 10-6-07. Albumen removed. No sugar. Many crystals were found which dissolved in from 5 to 7 seconds. Control—no crystals. Laboratory diagnosis—pancreatitis. Clinical history:—Attacks simulating gall stone colic for two or three weeks. Very tender in epigastrium and over gall bladder. Vomiting and a very obstinate constipation. Patient was sent in with a diagnosis of gall stones. When seen at hospital there was a temperature of $102\frac{1}{2}$ and a general peritonitis. At operation general peritonitis, fat necrosis and a gangrenous pancreas were found but the gall bladder was normal. Death ensued in 24 hours. No subsequent examination was possible.

Case 2. Urine from case 1 of first series. Few crystals soluble in 10 to 20 seconds. Patient is now in good health.

Case 3. Mr. L. 2-5-08. No sugar nor albumen. Many crystals were found which were soluble in 10 to 15 seconds. Control—no crystals. Laboratory diagnosis—pancreatitis. Clinical history:—Patient was operated on several years ago for cholecystitis. Adhesions about gall bladder but no stones.

Since operation there have been occasional attacks of vomiting and putty colored stools for 2 or 3 days. Some troublesome constipation.

Case 4. Mrs. F. 4-16-08. Albumen removed. No sugar. Many fine crystals soluble in 5 seconds. Control—no crystals. Laboratory diagnosis:—pancreatitis. Clinical history:—Attacks simulating gall stones for several months.

Case 5. Mrs. M. 4-23-08. First sample 48 hours old gave no reaction. Fresh sample 4-26-06. No sugar nor albumen. Many fine crystals soluble in 10 to 12 seconds. Control—no crystals. Laboratory diagnosis—pancreatitis. Clinical history:—Patient was operated on a year ago for gall stones. Present trouble began with severe pain and tenderness in epigastrium and profound prostration. Putty colored stools and constipation. Clinical diagnosis:—gall stones and pancreatitis. Operation revealed an enlarged gall bladder, a stone in the ampulla of Vater and an enlarged pancreas which was very hard. No post mortem could be obtained.

In my work with this method of examination I have never obtained a positive result except in cases where the pancreas was under suspicion or the biliary tract was the seat of an infection. The diagnosis of disease of the pancreas was confirmed in every case in which a direct examination of the gland could be made.

If further observations indicate, as now appears probable, that this examination will give positive evidence of disease of the pancreas it is of great value, not alone in primary disease of this gland but also as an aid to diagnosis of disease of the biliary tract.

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OBSERVATIONS ON CARBOLIC ACID AS A DISINFECTANT WHEN MIXED WITH ALCOHOL AND GLYCERIN.

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Carbolic acid, (phenol) has been used as a disinfectant and antiseptic for many years—in fact before bacteriology became an exact science. It probably owed its popularity as much to its odor as to the beneficial results obtained by its use. It is not the purpose of this paper to reestablish its claim as one of our most valuable disinfectants. Such is too well known to require any further discussion. Rather is it the object to correct some of the abuses to which it has been subjected.

It has for some time been known that alcohol would neutralize the caustic effects of phenol, when the latter was applied to the skin or mucous surfaces. This led many practitioners to use phenol in strong solutions, to limit such infective processes as diphtheria—following its application by the use of alcohol, thus preventing an undue escharotic effect on the mucous membrane of the throat. This method is in itself commendable, but some have adopted the plan of mixing the alcohol and phenol before applying it to the infected surface, believing that by so doing they obtain the same results as when the two reagents are used separately.

To carry out any series of tests, the value of the method used must not be underrated. To this end several experiments were carried on simultaneously to determine which of the three most popular methods (Koch's, Hill's or Rideal-Walker's) was most applicable to the work in hand. Koch's method which is the oldest is as follows:— Small pieces of silk thread are soaked in a bouillon culture of a given organism, and dried at a temperature not exceeding 38° C. These are then placed in the disinfectant solution and at definite intervals of time are removed to sterile tubes of bouillon with

sterile forceps, then incubated at a given temperature for a sufficient length of time. The faults of this method are the difficulty in getting the threads equally inoculated, drying them at the proper temperature, and transferring them from one tube to another without contamination; its greatest fault lies in the fact that the results are not reliable and can not be depended upon, due no doubt to the harmful influence of drying. A glance at tables I and III carried out under conditions exactly similar, will prove this statement. Hill's method consists of drying the organism on the ends of sterile glass rods, transferring these to the disinfectant solution, thence to sterile tubes of broth, much the same as in Koch's method. The difficulties met here are almost identical with those before cited, that of drying, inoculation, etc. Tables II and V representing experiments made under the same conditions indicate that this method is also unreliable.

In 1903 Rideal and Walker developed an easy and ingenious method for the standardization of disinfectants, which modified to meet our conditions, is as follows: A test tube rack having an upper and lower row of holes is used. In the upper row are placed two test tubes containing certain dilutions of the disinfectant, in the lower row ten tubes of sterile bouillon. Five drops of a 24 hour culture of the organism in bouillon are added to the first tube of disinfectant, which contains 5 c. c.; after 2 minutes, the same amount is added to the second tube of disinfectant. From these sub-inoculations are made into the lower tubes at intervals of one minute by means of a sterile platinum loop, giving us five sub-inoculations from each tube, in which the disinfectant has acted for 1, 2, 3, 4, and 5 minutes respectively. These sub-inoculations are incubated at 37° C for 72 hours and growth noted. With each series a control is run, made by transferring a loopful of the original bouillon culture to a tube of bouillon, to make certain that the original culture was in good condition. To determine whether or not the disinfectant, carried over in the loopful of bouillon, when the sub-inoculations are made, has any inhibitory effect on the growths, a loopful of the particular dilution of the disinfectant is always transferred to the control. All of our observations indicated that the growth in the control was unaffected.

To make tests reliable, the conditions governing them must be kept uniform, hence the bouillon was always made to give a reaction of +1 to phenolphthalein. The culture, which in all the tests was *Bac. typhosus*, was always used when 24 hours old,

since cultures of varying ages have different powers of resistance as have also different strains of the same organism, hence one strain was used throughout. Incubation was always carried on at 37°C. and prolonged for 72 hours. The room temperature when making the inoculations should also be kept uniform, since various temperatures will give various results. A rise in temperature of a very few degrees will often cause a great difference in the bactericidal action of a disinfectant. This fact explains the apparent discrepancy in results shown by tables III and IX, the latter test being carried out on a much warmer day than the former, hence the greater power of the disinfectant, and the absence of growth in the tubes. After the three methods were thoroughly tested the Rideal-Walker method was chosen because of its greater accuracy, ease, and speed of manipulation for the real purposes of the experiment. Alcohol was tested in solutions ranging from 1 to 5% and found to have scarcely any bactericidal action in such dilutions, as shown by table VII. Glycerin was tested in the same manner and found to have even less value as a disinfectant. To make conditions absolutely uniform, phenol was tested under the same conditions each time as the others, table IX.

After having tested the disinfecting power of alcohol and phenol, separately, they were mixed in solutions which contained 1% of phenol and 1% of alcohol, 2% of phenol and 2% of alcohol, and so on up to 5% of each. Table X tells the story. The disinfectant action of the carbolic acid was entirely destroyed by mixing it with the alcohol. It is to be understood that each table given is chosen from several, and taken to show a typical result.

In table XI glycerin was mixed with the phenol in the same manner as above. A comparison of this with table XII which is for unmixed phenol, shows that if there is any neutralization of the disinfectant power of phenol, it is extremely slight. Glycerin will not neutralize the caustic action of phenol to any great extent when applied to the skin nor does it appreciably reduce the disinfectant value of phenol. Alcohol immediately neutralizes the caustic action of phenol, and also destroys entirely its bactericidal action. (Table X)

From the above data we must conclude that the property of phenol which gives to it its caustic action, also accounts for its disinfectant action, since neutralization of the one destroys the other. The nature of the chemical reaction which takes place

when the two are mixed is not well understood nor is the manner in which phenol acts on the bacterial cell to produce its death. It is probably a coagulation process of the microprotein of the cell, without, however, a chemical combination as is found in disinfection with mercuric bichloride. At any rate we have shown that whatever this process may be, it is totally destroyed when alcohol is mixed with it, the result being an inert compound, which is worse than useless, as it gives a sense of false security.

The following tables are based on experiments with the 24 hour old bouillon culture of the *Bacillus typhosus*. After the exposure to the disinfectant the tubes were incubated at 37° C. for 72 hours. A control was always made.

I

KOCH'S METHOD

Disinfectant			1'	2'	3'	4'	5'
Carbolic Acid	1 %		—	—	—	—	—
" "	2 %		—	—	—	—	—
" "	3 %		—	—	—	—	—
" "	4 %		—	—	—	—	—
" "	5 %		—	—	—	—	—
Control +							

II

HILL'S METHOD

Disinfectant			1'	2'	3'	4'	5'
Carbolic Acid	1 %		—	—	—	—	—
" "	2 %		—	—	—	—	—
" "	3 %		—	—	—	—	—
" "	4 %		—	—	—	—	—
" "	5 %		—	—	—	—	—
Control +							

III

RIDEAL-WALKER METHOD

Disinfectant			1'	2'	3'	4'	5'
Carbolic Acid	1 %		+	+	+	+	+
" "	2 %		+	+	+	+	—
" "	3 %		—	—	—	—	—
" "	4 %		—	—	—	—	—
" "	5 %		—	—	—	—	—
Control +							

IV

KOCH'S METHOD

Disinfectant			1'	2'	3'	4'	5'
Carbolic Acid	1 %		+	+	+	—	+
" "	2 %		—	—	—	—	—
" "	3 %		—	—	—	—	—
" "	4 %		—	—	—	—	—
" "	5 %		—	—	—	—	—
Control +							

V

HILL'S METHOD

Disinfectant			1'	2'	3'	4'	5'
Carbolic Acid	1 %		+	+	+	+	+
" "	2 %		+	—	+	—	—
" "	3 %		—	—	—	—	—
" "	4 %		—	—	—	—	—
" "	5 %		—	—	—	—	—
Control +							

VI

RIDEAL-WALKER METHOD

Disinfectant			1'	2'	3'	4'	5'
Carbolic Acid	1 %		+	+	+	+	+
"	2 %		+	—	+	+	—
"	3 %		—	—	—	—	—
"	4 %		—	—	—	—	—
"	5 %		—	—	—	—	—
Control +							

VII

Disinfectant			1'	2'	3'	4'	5'
Alcohol	1%		+	+	+	+	+
"	2%		+	+	+	+	+
"	3%		+	+	+	+	+
"	4%		+	+	+	+	+
"	5%		+	+	—	—	—
Control +							

VIII

Disinfectant			1'	2'	3'	4'	5'
Glycerin	1%		+	+	+	+	+
"	2%		+	+	+	+	+
"	3%		+	+	+	+	+
"	4%		+	+	+	+	+
"	5%		+	+	+	+	+
Control +							

IX

Disinfectant			1'	2'	3'	4'	5'
Carbolic Acid	1 %		+	+	—	—	—
"	2 %		—	—	—	—	—
"	3 %		—	—	—	—	—
"	4 %		—	—	—	—	—
"	5 %		—	—	—	—	—
Control +							

X

Disinfectant					1'	2'	3'	4'	5'
Equal Volumes of Carbolic Acid and Alcohol 1% each					+	+	+	+	+
"	"	"	"	2%	+	+	+	+	+
"	"	"	"	3%	+	+	+	+	+
"	"	"	"	4%	+	+	+	+	+
"	"	"	"	5%	+	+	+	+	+
Control +									

XI

Disinfectant					1'	2'	3'	4'	5'
Equal Volumes of Carbolic and Glycerin 1% each					+	+	+	+	—
"	"	"	"	2%	—	—	—	—	—
"	"	"	"	3%	—	—	—	—	—
"	"	"	"	4%	—	—	—	—	—
"	"	"	"	5%	—	—	—	—	—
Control +									

XII

Disinfectant					1'	2'	3'	4'	5'
Carbolic Acid	1	%			+	+	+	—	—
"	"	2	%		—	—	—	—	—
"	"	3	%		—	—	—	—	—
"	"	4	%		—	—	—	—	—
"	"	5	%		—	—	—	—	—
Control +									

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PSEUDO-PARASITES

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INTRODUCTION:—During the last few years much attention has been paid to the clinical diagnosis, both chemical and microscopical, of the fæces and the parasites found therein. The subject of parasites has become a study of itself and much investigation has been made on parasites affecting the human being by Leuckart, Stiles, Ward and others.

It was while making the investigation of the different parasites that the presence of pseudo-parasites in the human being was discovered. I have endeavored to review the literature on the subject, and to record the observations made in the laboratory while studying the pseudo-parasites found in man.

In order to determine the structure and nature of pseudo-parasites I have studied a number of plants and other substances, both by gross and microscopical examination of the raw material and by experiments consisting of the ingestion of such substances and the subsequent examination of fæces.

The accompanying drawings represent various structures which have been mistaken for real parasites.

TECHNIQUE:—The experiments consisted of large quantities of different substances and by subsequently examining the stools and finding the material which represented the undigested portion of the material ingested. For these experiments the following substances were used:—bananas, lemons, sauer kraut, onions, apples, thread, rhubarb, oranges, celery, dates, oatmeal and pineapple.

To best bring out the particular structure of the different substances used, sections of suitable length were taken and first exposed to the action of the air for a variable length of time—at least a half day. They were then placed in a five per cent solution of potassium hydrate for one day. This

treatment tends to separate those elements which usually resist digestion from the remaining digestible substances. The action of the light and the potassium hydrate caused the undigestible elements to assume a dark color similar to those discharged from the bowels. The dark staining indigestible elements were then further separated by means of needles and placed in a separate dish containing five per cent potassium hydrate. In most cases these dark colored, indigestible elements consisted of fiber bundles, as will be shown later. The fiber bundles were divested of all the softer digestible part and were then ready for mounting, either in glycerin or balsam. Glycerin tends to clear the specimen, and is used for that reason. When it was desired to make a permanent mount they were clarified in glycerin for twenty to thirty minutes, then mounted in balsam. These sections were then kept for future reference for comparison with those obtained from the discharge from the bowels.

As experimental work, I ingested large quantities of the different fruits and vegetables, making the diet for the day consist principally of one article. At various intervals following the ingestion of the different substances examinations of the stools were made, and especially after the end of twenty-four hours, this being the time generally required for the food to pass the length of the intestinal tract. A better rule is to take the second stool following the ingestion of the substance. The stools were collected in glass jars with a tightly fitting top, and a four per cent solution of formaldehyde was added. This readily deodorizes and softens the stools although if left too long in formalin they become hardened. The color, odor and consistency were noted before the addition of the four per cent formaldehyde.

Suspicious looking particles of the fæces were spread out on a slide and covered with a large cover glass. Large slides are best adapted for this purpose, the fæces being spread out in a very thin layer. When an examination of all substances is desired, I have found that a very good way is to make an emulsion of the fæces and then allow the mixture to settle and dry on the slide. If a thin watery emulsion is made it was found that coarse vegetable remnants, cells and crystals formed the lower layer, parasites and eggs the middle, while in the upper layers were the bacteria, fat, vegetable cells, etc. These can be drawn up with a pipette and placed on a slide, spread out thin and covered with a large cover glass. The slides are

then examined under the two-thirds and one-sixth objective of the microscope, the higher power being used to bring out the detailed characteristics. These were then compared with the specimens prepared by treatment with potassium hydrate and identified as the same.

PSEUDO-PARASITES—Pseudo-parasites are divided into two classes:

First. Those which are actually free, living animals introduced by accident, usually in food and drink, into the human alimentary canal, and exciting therein abnormal conditions which induce their more or less immediate forceful expulsion. Examples: Botkin found in the vomit of a Russian, numbers of small nematodes, which he wrongly thought to be a human parasite. This nematode lives normally in the onion and its introduction into the stomach with this vegetable excited the symptoms noted. Cases of *gordius*, the hair snake:—About a dozen cases taken from man after a supposed sojourn of a few hours to fourteen days have been reported. Some of these have been vomited, others passed per anum. This form has often been mistaken by the physician for a true parasite, and in one celebrated case, at least, as a guinea worm. There are forty recorded cases where *mysiopodes* were mistaken for pseudo-parasites. The ingestion of such forms is purely accidental. The symptoms are those of helminthiasis. They remain at longest a very short time and never show adaptation to their new environment.

Second. This class is composed of parts of substances of both plant and animal origin, used as food, which have not been destroyed by the action of the digestive juices. Examples: The radulae of the common limpet have on several occasions been reported as found in the stools. The seeds of the mulberry have been mistaken for a parvalin worm, and plant tissue and other similar undigested structures of peculiar shape, appearing periodically, as new helminthes. That a differentiation of such structures is not easy is proved by the account given by Stiles and Bierring and Albert of the similarity of appearance of the partially digested banana fibers to minute tapeworms. Some years ago Leuchart entrapped a group of students doing research work in helminthology with the pulp

vesicles of an orange which were found in the field of examination.

PSEUDO-PARASITES SIMULATING ANIMAL PARASITES.

BANANA:—The fibers of banana have been mistaken for tapeworms. C. W. Stiles, was probably the first to make an investigation of the subject. Specimens were submitted to him for examination, and only after calling in the aid of a botanist, was he able to determine their nature. In our own laboratory during the winter of 1902-'03 attention was directed in two instances to material discharged from the bowels that had a marked resemblance to minute tapeworms, and led to considerable and interesting study before its real nature was finally determined. The objects in question appeared in considerable numbers, were dark brown or black and seemed to be segmented like a tapeworm. Experiments made by Doctor Albert exposing transverse sections of bananas to the light revealed the presence of dark colored fibers or bands along the center and on the external surface. (See figure.) These were brought out more distinctly by placing sections in a five per cent solution of potassium hydrate. The segmented, band-like elements, which usually resist digestion separated from the remaining substance of the banana and assumed the same appearance as those discharged from the bowel. The arrangement of these fibers divides the banana into three well defined portions. In order to fully establish the identity of the bodies described personal experiments consisting of eating large quantities of bananas and subsequently examining the stools were made. Microscopically, the fibers were found to be made up of small oblong bodies or segments, apparently more or less connected and resembling tape worm strobilæ. When more closely examined they are found to consist of bands or rows of cells oblong in shape, well defined in outline, composed of a granular material with occasional openings or spaces, evidently due to retraction of the protoplasm. The cells seem to overlap each other at the ends and so form an apparent connection with each other. These represent the parenchyma cells. The spiral elements seen in the figures, are the so-called spiral ducts—fibro-vascular bundles or trachæ—of the banana. Even when magnified the oblong-shaped cells bear a striking resemblance in form, shape and arrangement to the segments of a tapeworm. They might be mistaken for the *Tænia nana* or the *Tænia diminuta*.

Of the two cases to which the attention of this laboratory has been called, one was from a child two years of age who had

been passing material of this kind for some time. According to the mother, "the child became more or less restless just before passing these false tapeworms". The second case was that of a man thirty-eight years of age, suffering with anæmia of obscure origin, in whose stools these pseudo-parasites had been occasionally observed. Upon further correspondence with the attending physicians the connection with the ingestion of banana was fairly well established in both instances.

Differential diagnosis between banana pseudo-parasites and:—

(a) *Tænia nana*, the body of which is filiform, gradually tapering into the neck; head obtuse anteriorly, with almost globular suckers and pyriform rostellum armed with a crown of bifid hooks. It is from five to forty-nine mm. in breadth, and has about one hundred and fifty to two hundred segments.

(b) *Tænia diminuta*, *Hymanolipis diminuta* (Rudolphi) whose heads are conical, suckers situated anteriorly, neck long and slender; segments with obtuse angles, length always remaining greater than their width. Worms 100 mm. long, 1 to 4 [mm. broad.

ORANGES:—In the orange we find structures that bear a resemblance to certain trematodes. The orange is made up of a great number of small cells lanceolate to oblong-oval in shape, size from 15 to 30 mm. long and 4 to 8 mm. wide. These are best brought out by separating the orange into its different sections or carpels and removing from the section the transparent covering which seems to hold these many pulp cells together. Each section will be found to consist of a large number of pulp cells compactly arranged. One of these is placed in a 5 % solution of potassium hydrate or plain water and readily separated with needles into the separate cells which are nearly transparent. Ingestion of oranges in large numbers and subsequent examination of the fæces did not reveal the presence of these cells but upon examination of the stomach contents these cells were found. They very much resembled the liver fluke, *Opisthorchis sinensis*, *Opisthorchis noverca* and the *Fasciola hepatica*. Lueckart has reported a case of orange cells being mistaken for a parasite. Stiles reports pieces of partially digested orange sent in as liver flukes by physicians, pathologists and boards of health.

One case has come to my knowledge through Dr. Linn of Des Moines. A man of middle age and in apparently good

health brought to his office a portion of fæces which he said he had passed and had noticed the same condition on previous occasions when troubled with spells of indigestion. The Doctor made an examination of the fæces, found these portions which resembled worms and upon examining them with a low power of the microscope was puzzled to ascertain their nature. The patient brought similar specimens to the doctor's office several times. On questioning the patient he found that these supposed worms occurred in the fæces only after eating oranges, and that oranges did not agree with him. The doctor was then able to determine the nature of the pseudo-parasite. Another case reported at the university is that of a man taken with sudden and profuse vomiting lasting for several hours, with great prostration but subsequent recovery. In the examination of the vomitus, bodies resembling flukes were discovered. The specimen was referred to Professor Macbride, Department of Botany, who decided that they were undoubtedly orange cells.

LEMON:—The lemon is very similar in structure to the orange, and differs only in the arrangement of the cell contents, which can readily be distinguished by the use of a microscope. These cells can readily be obtained by cutting the lemon, open and taking out a section. If placed in potassium hydrate or water they can readily be separated into the individual cells, which are from 10 to 25 mm. long, and 4 to 8 mm. broad. They are lanceolate in shape and provided with structures resembling a pointed head and a tail, which varies in length or may be absent. They are nearly transparent, and if separated, are nearly flat. These cells, like those of the orange, macroscopically resemble the *Opisthorchis noverca*, *Fasciola hepatica* and *Opisthorchis sinensis*. It can be differentiated from them, however, by examination under the one-sixth power of the microscope, it being composed of a double cell wall with the cell structure inside. It contains no mouth or oral sucker nor intestines.

On the other hand the *Opisthorchis sinensis* is 10 to 20 mm. long and 2 to 5 mm. broad; its oral suckers are larger than the ventral acetabulum, which is on the border of the first and second parts of the body. Skin has no spines, pharynx globular, 22 mm., in diameter. Oesophagus 17 mm. long. Bifurcation of intestines nearer to oral sucker than acetabulum. The *Opisthorchis noverca* is 9 to 12.5 mm. long and 2 to 5 mm. broad. Oral suckers terminal; ventral acetabulum much smaller than oral suckers; distance between two equal to about

one-eighth of the length of body. Skin thickly beset with spines. Oesophagus apparently absent.

The *Fasciola hepatica* is 18 to 51 mm. long and 4 to 13 mm. broad. Oral sucker 1 mm. in diameter. Ventral acetabulum about 1 to 6 mm. wide. Spines on skin arranged side by side in alternate rows. Bifurcation of the intestines is immediately anterior to genital pore.

CELERY:—The part of the celery which may be mistaken for animal parasite is found in the fibro-vascular bundles, which can be readily seen in a cross section of the stalk. In experimenting with the celery, it was first masticated, then placed in a glass dish and exposed to the light following which it was treated with potassium hydrate, which tended to separate the resisting band-like elements from the remaining soft cells, causing them to assume the same dark color as when found in the fæces. It was found that the fibro-vascular bundles extend vertically and run the length of the stalk. They are seen scattered at irregular intervals, mostly at the periphery, and have a yellowish color in the fresh preparation. Large quantities of celery were also eaten. On subsequent macroscopic examination of the stools, which were of light yellow, pasty character, long yellowish white shreds resembling worms were found. They were from 10 mm. to 100 mm. in length, the average being 20 to 75 mm. long, and 2 to 3 mm. broad. They were yellowish in appearance but on short exposure to the air turned much darker. The length of these is due to the fact that when eating celery, when one takes a bite he draws these fibro-vascular bundles out to quite an extent before they break off. Macroscopically these celery shreds greatly resemble the *Ascaris mystax*, *Oxyuris vermicularis*, *Uncinaria Americana* (Stiles), *Trichocephalus trichuirus*, *Diphylidium canium*. Single fibers may under the microscope resemble the *Tænia nana* and *T. diminuta* or segments of others. So it can readily be seen how, if a patient complained of pain, tenesmus, and especially a violent tickling about the anus and its neighborhood, excitation of the sexual organs, etc., and finding these fibro-vascular bundles of celery which so closely resemble the different parasites named, a mistake in diagnosis could readily be made.

Several cases of celery fibers having been mistaken for parasites have come to my notice. They were in children between the age of one and two years. The fibers were brought to a physician's office by the parents, having been found in the

stools after an attack of indigestion. They were about the size of pin worms. Upon examination by the physician he was much puzzled to determine their character. Testing them for cellulose I at once established their vegetable nature. Upon inquiry it was found that the children had been given a stalk of celery and other articles of food at a Sunday dinner which had caused the spell of indigestion and the presence of the celery fibers in the fæces.

Celery fibers must be distinguished from the (a) *Ascaris mystax* which is 45 to 60 mm. in length. The head is pointed and provided with wing-like projections. It has three lips which are provided with suckers. It is cylindrical in shape (b) *Oxyuris vermicularis*, which is 10 mm. long. On the head are three lip-like projections with lateral cuticular thickening. The tail has six pair of papillæ. (c) *Uncinaria Americana*, 12 to 18 mm. long. Body cylindrical; mouth cavity is provided with two pair of semilunar plates instead of ventral teeth. A dorsal conical tooth projects prominently into the buccal cavity. Vulva situated in front of posterior third of body. (d) *Trichocephalus trichiurus*, 40 to 50 mm. in length. On each pole is a projection which is lighter than the dark brown shell. (e) *Dipylidium caninum*, 35 mm. long, 3 mm. wide, head is small, somewhat rhomboidal in shape with a projecting rostellum which bears three or four circles of hooks. The proglotoids are 6 to 7 mm. in length and sausage-shaped with the genital pores symmetrically arranged on either side.

ONIONS:—The onion also affords a chance for mistaken diagnosis. For experiment I used the common red onion (*Alium cepa*). In a cross section of an onion dark portions of a yellowish color and more dense structure are found. It is these parts which are least destroyed, if an onion be cut up, exposed to the air, and, later, placed in potassium hydrate solution of five per cent. Near the center and between the different layers will be found what are undoubtedly the roots and other parts, yellow in color which no doubt become the stake. These parts are also but slightly acted upon by air and potassium hydrate. The onions were taken raw, cut into sections—quite a quantity being eaten at one time. Upon subsequent examination of stools, large fibers resembling worms and parts of the outer skin layer composed of parenchyma cells were found. These fibers of the onion were whitish in color but stained slightly by the stools, which were yellowish brown in

color and pasty in consistency. In length they were from 40 to 100 mm., and in breadth 3 to 4 mm., tapering at both ends. The small fibers may resemble the *Ascaris mystax* or *Anchylostoma duodenale*. The presence of these fibers, together with the symptoms similar to those caused by the nematodes, namely, disturbance of the gastric-intestinal tract, disturbances of nutrition and nervous symptoms, make a correct diagnosis difficult without the aid of the microscope. Upon microscopical examination the onion fibers are found composed of fibro-vascular bundles, all parallel to each other. The contour of the cell walls can readily be seen, and they generally give the reaction for cellulose.

Pieces of onion may be mistaken for the (a) *Ascaris lumbricoides* which has a head consisting of three projections or lips provided with suckers. The tail end of the male is rolled up on its ventral surface like a hook, and is provided with papillæ. (b) *Anchylostomum duodenale*, (Dublini). Length 10 to 18 mm. Its head is turned towards its back. The mouth capsule is surrounded by four teeth. The tail of the male forms a three-lobed bursa. (c) *Ascaris mystax*, already described.

SAUER KRAUT: This is a very common article of diet, especially among the German, Dutch and Bohemian population of our country. I found on taking the kraut as found in the stores, exposing it to the air and potassium hydrate, that it turned a slightly yellow color, and that the thick white portions were little affected. I made mounts of the thinner portions, which were easily affected by the treatment. This kraut had evidently been cut by a machine, which left it in long shreds. Upon microscopical examination of these mounts, the structure of the kraut was seen to consist of cells running parallel, while in places spots, yellow in color and composed of granular material, were seen.

In experimenting I ate a large quantity of the kraut, chewing some portions more than others. The kraut used was from the same supply used in the making of mounts. Examining the fæces microscopically, I found that the shreds of kraut, little affected by digestion, resembled some of the parasites. These shreds were about 1 to 5 mm. wide and 25 to 100 mm. long. They were quite white in appearance, being stained but slightly by the fæces. They closely resembled a

worm and might be mistaken for the undeveloped *Ascaris lumbricoides*, *Ascaris mystax* or *Uncinaria Americana*.

DATES:—With dates the same method was pursued as already described. The parts which may be mistaken for parasites are to be found in the thick whitish covering of the seed (sclerenchyma cells), fibro-vascular bundles and the skin. A date was taken and the pulp separated from the skin. The skin was placed in potassium hydrate, five percent. The pulp was also separated from the seed. The covering of the seed consisting of sclerenchyma cells was also placed in potassium hydrate and all pulp removed. This was then teased out with needles into small white bands, which, when examined with the microscope, were seen to consist of fibro-vascular bundles. For the experimental part I ate a large number of dates, and subsequently examined my stools. Upon macroscopic examination the white tough bands or portions of the skin covering the seeds were found. These varied in size from 20 to 30 mm. long, and from one to several cells wide. These fiber-like portions of the skin, when examined under the microscope, were found to consist of sclerenchyma cells running parallel to each other and slightly darkened. Upon placing a thin film on a slide I found many of the single fibro-vascular bundles, which showed that these had become separated in passing through the gastro-intestinal tract. The larger portion closely resembled the segments of tape worms, the arrangement of the fibro-vascular bundles being much like that of the segments.

OATMEAL:—This I thought ought to be mentioned, as a case was brought to my knowledge in which the shell of the oat after passing through the digestive tract was mistaken for the segments of worms. A large quantity of oatmeal as ordinarily cooked was eaten and examination of the stool made. There was readily visible in the stool small white particles, soft in consistency and closely resembling segments of a tapeworm. They were from 1 to 4 mm. long and from .5 to 1.5 mm. wide. Many were elliptical in shape, while others were rectangular. These might readily be mistaken for segments of the tæniae or their eggs. However, upon microscopical examination their vegetable character was readily seen in the cell arrangement, and this with the test for cellulose was found sufficient for a differential diagnosis.

THREAD: Pieces of common white sewing thread was mixed with food and swallowed. These threads appeared in the

stool but little altered, being only slightly discolored by the bowel contents. Many were obtainable with a needle without washing being readily visible in the fæces as passed and very closely resembling worms. The coarse thread, upon careful microscopical examination, could readily be distinguished from worms or parasites because of their division at the ends. The finer ones I found to closely resemble the round worms:—*Oxyuris vermicularis*, *Ascaris mystax*, *Dipylidium canium*, *Trichocephalus*, *Trichiurus*, and *Uncinaria duodenale*.

Undoubtedly women and tailors may be found to be affected with this pseudo-parasite. In pulling out basting threads or biting off the ends after tying a knot, they often swallow these portions of thread, so that their presence in the fæces might lead to a mistaken diagnosis.

RHUBARB:—The stalk of the rhubarb is quite similar to that of the celery, and is composed of parenchyma cells, among which are the fibro-vascular bundles. A cross section shows the number and arrangement of the fibro-vascular bundles, which contain from six to eight ducts.

I took a stalk of rhubarb, peeled it and exposed it to the action of light, air and a five per cent solution of potassium hydrate, to better bring out the fibro-vascular bundles. The bundles resisted the action of light, air and potassium hydrate more than did the arenchyma cells, which were separated and partially destroyed. I ingested a large quantity of the cooked rhubarb and subsequently examined my stool; in which I found many shreds of dirty white color. The macroscopical appearance of these markedly resembled some of the worms. The shreds resembling worms were 10 to 20 mm. in length and 2 to three mm. wide. These, after being washed, were examined under the microscope and found to consist of fibro-vascular bundles with a few attached parenchyma cells. These shreds of the rhubarb much resembled, in macroscopical appearance, the *Oxyuris vermicularis*, *Uncinaria Americana* (Stiles), *Uncinaria duodenale*, and the *Trichocephalus trichinus*.

PINEAPPLE:—The pineapple is another fruit with which experiments were made. To determine the gross and microscopical appearance of the raw material, a longitudinal section was made. This showed it to be composed of parenchyma cells and fibro-vascular bundles, which run from the periphery toward the center, and then, ascending, formed the center or core. On being exposed to the air and placed in a five percent solu-

tion of potassium hydrate, these fibro-vascular bundles were readily seen. I separated them by the use of a needle from the softer and partially disintegrated parenchyma cells, and examined them under the microscope and found that the larger fibers consist of four or five fibro-vascular bundles surrounded by one or two layers of parenchyma cells arranged parallel to each other. Large quantities of longitudinal sections of pineapple were ingested. Numerous shreds were found in the fæces upon subsequent examination. They were of a whitish color, and varied from 5 to 10 mm. in length, and from 1 to 3 mm. in width. In many cases the ends were curled up.

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(Plates with explanation follow)

EXPLANATION OF PLATE III

Figure No. 1

A Parenchyma cells of banana

B Fibro-vascular bundles of banana

Figure No. 2 Banana—spiral ducts of fibro-vascular bundles magnified.

Figure No. 3 Cross section of Banana

Figure No. 4 Banana shreds

Figure No. 5 *Tænia nana*

Figure No. 6 *Tænia nana*, magnified

Figure No. 7 Banana spiral ducts, enlarged

Figure No. 8 *Tænia nana*, enlarged

Figure No. 9 *Oxyuris vermicularis*

Figure No. 10 Date fibres

Figure No. 11 *Uncinaria duodenale*

Figure No. 12 *Oxyuris vermicularis*, magnified

Figure No. 13 Date fibre, magnified

Figure No. 14 *Uncinaria duodenale*, magnified

PLATE III



Fig I



Fig II

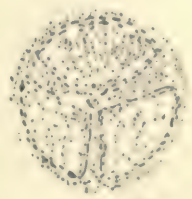


Fig III



Fig IV



Fig V



Fig VI



Fig VII



Fig VIII

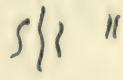


Fig IX



Fig X



Fig XI



Fig XII



Fig XIII



Fig XIV

EXPLANATION OF PLATE IV

- Figure No. 1 Orange cells
- Figure No. 2 *Distoma conjunctum*
- Figure No. 3 *Distoma spatulatum*
- Figure No. 4 *Distoma hepatica*
- Figure No. 5 Orange cells, magnified
- Figure No. 6 *Distoma lanceolate*, magnified
- Figure No. 7 Lemon cells, magnified
- Figure No. 8 Lemon cells
- Figure No. 9 *Distoma spatulatum*, magnified

PLATE IV



Fig I



Fig II



Fig III



Fig IV



Fig V

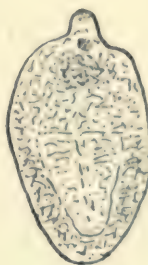


Fig VI



Fig VII

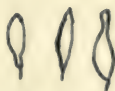


Fig VIII



Fig IX

EXPLANATION OF PLATE V

- Figure No. 1 Rhubarb fibres
- Figure No. 2 Cross section rhubarb
- Figure No. 3 *Trichocephalus trichorous*
- Figure No. 4 *Oxyuris vermicularis*
- Figure No. 5 Rhubarb fibres, enlarged
- Figure No. 6 *Anchylostomum duodenale*
- Figure No. 7 Pineapple fibres
- Figure No. 8 *Uncinaria duodenale*
- Figure No. 9 Pineapple fibres, enlarged
- Figure No. 10 Celery fibres
- Figure No. 11 *Ascaris mystax*
- Figure No. 12 Celery fibres enlarged

PLATE V

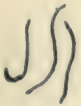


Fig I



Fig II

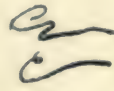


Fig III



Fig IV

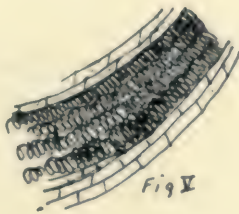


Fig V



Fig VI

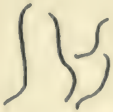


Fig VII



Fig VIII

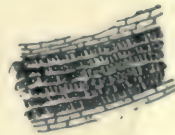


Fig IX



Fig X



Fig XI



Fig XII

EXPLANATION OF PLATE VI

- Figure No. 1 Sauer kraut fibres
- Figure No. 2 Ascaris mystax
- Figure No. 3 Sauer kraut fibres, magnified
- Figure No. 4 Ascaris lumbricoides
- Figure No. 5 Cross section onion
- Figure No. 6 Onion fibres
- Figure No. 7 Onion fibres, magnified
- Figure No. 8 Thread
- Figure No. 9 Thread, magnified
- Figure No. 10 Oatmeal

PLATE VI



Fig I



Fig II

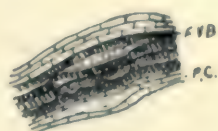


Fig III

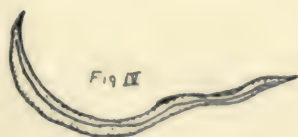


Fig IV

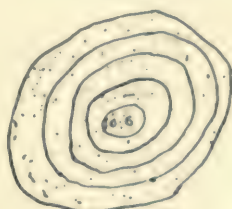


Fig V



Fig VI

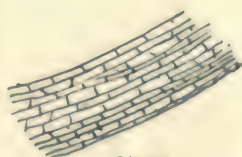


Fig VII

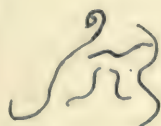


Fig VIII



Fig IX



Fig X

STATISTICAL DATA AND INTERPRETATION OF CERTAIN LABORATORY FINDINGS AND CLINICAL OBSERVATIONS IN DIPHTHERIA

(Based on 2660 examinations for diphtheria bacilli made in the Bacteriological Laboratory of the Iowa State Board of Health during the year July 1, 1907 to July 1, 1908).

HENRY ALBERT, M. D., IOWA CITY, IOWA

Director Iowa State Board of Health Bacteriological Laboratory

Every diphtheria specimen sent to the Laboratory is accompanied by a card which has been filled out by the physician sending same and which presents certain clinical data relative to the case in question. The result of the bacteriological examination is later recorded on the same card. From these cards the following interesting and instructive tables have been compiled. They are based on 2660 examinations, of which 1140 were for diagnosis (Tables 1-10) and 1520 for release from quarantine (Table 11). Table Nos. 1 and 10 are based on all specimens sent for diagnosis; tables 2-9 inclusive are based only on those specimens for diagnosis in which diphtheria bacilli are found; tables, 5, 6, 7, 8, and 9 represent data as given when the specimen for diagnosis was submitted. Such data may of course have changed during the course of the disease. Only one culture was submitted for diagnosis from 1112 patients, two cultures from 28 patients; Of those which proved to be positive, 145 were males, 199 females; 41 were married, 182 unmarried and 4 widowed; 51 were from patients 1 to 5 years old; 148 from 6 to 15 years, 118 from 16 to 60 years and 5 from patients over 60 years of age.

TABLE I

(Diagnosis by Physician and Laboratory Findings).

A Diphtheria (by physician)	
Laboratory found diphtheria bacilli.....	230
Laboratory did not find diphtheria bacilli.....	64
B Not diphtheria (by physician)	
Laboratory found diphtheria bacilli.....	27

	Laboratory did not find diphtheria bacilli	74
C	Possibly diphtheria (by physician)	
	Laboratory found diphtheria bacilli	16
	Laboratory did not find diphtheria bacilli	13
D	Uncertainty (by physician)	
	Laboratory found diphtheria bacilli	18
	Laboratory did not find diphtheria bacilli	11

This table is very interesting and instructive. It shows that diphtheria bacilli were found in 78% of cases in which the clinical diagnosis of diphtheria was made; in 26.7% of cases in which the clinical diagnosis of a non-diphtheritic inflammation of the tonsils, pharynx, larynx or nose was made; in 55% of cases where the physician made a diagnosis of "possibly diphtheria"; and in 64.2% of cases where the diagnosis was "questionable" in the minds of the physician. It is especially interesting to know that during the course of the past year the laboratory found diphtheria bacilli 78% of all cases diagnosed by the attending physician as diphtheria. The records for the first two years indicate that the percentage at that time was only 60%. The higher percentage given in our present table may be accounted for by the fact that physicians have been more careful in the making of their clinical diagnosis, especially by not calling every pseudo-membraneous inflammation of the throat diphtheria as was more true of our former records. This more conservative attitude on the part of the physician in making a positive diagnosis of diphtheria has also led to our finding the diphtheria bacilli in a higher percentage of the cases diagnosed by the physician as "not diphtheria"; "possibly diphtheria" and "uncertain" than was formerly the case. It does not mean, however, that the physician because of being more conservative has been less cautious as regards the care of the patient or its bearing on the public health as is suggested by the fact that according to the present tables antitoxin was given in 43.4% of all cases (which proved to be diphtheria) before the specimen was sent for diagnosis. According to the statistics based upon the examinations made from July 1, 1904 to July 1, 1906 antitoxin had been given to only 32.8% of the cases before the time the specimen was submitted for diagnosis. In this connection also it is of interest to note that according to the figures of the Department of Health of the city of Chicago as given in the biennial report for the year 1904-1905, diphtheria bacilli were found in only 37% of cases that were clinically diagnosed as diphtheria; in 16% of

cases clinically diagnosed as not diphtheria, and in 27% in which the clinical diagnosis was not given.

TABLE II
Occupation

Barbers	2
Business (indoors)	14
Housekeeper	33
Laborers (outside)	26
Physicians	1
Pupils and students	105
Teachers	9

This table brings out the fact that 105 or 54.4% of the cases in which diphtheria bacilli were found occurred among school children or students and that nine occurred among teachers. These figures together with the data of table No. IV indicate the important part which the school room plays in the transmission of diphtheria. The recent observations with reference to "bacillus carriers," individuals who, after recovery from the disease, carry in their nose, mouth and throat, virulent germs of the affection for a long time, and during that entire time are capable of transmitting such to others, should be well recognized by physicians. "Bacillus carriers" are not only those who have had the disease but also individuals who have been exposed to the germs but who have never contracted the disease. It has been found that during epidemics of the disease up to five per cent of the generally healthy public carry the germs in the nose or mouth. This all emphasizes the great importance of not releasing any person from quarantine so long as they are the bearers of virulent diphtheria bacilli as determined by bacteriological examinations. Two consecutive negative reports should be obtained. So long as such is not done, and that it is not done is evidenced by table No. 11, we can scarcely hope to stamp out diphtheria for a long time to come.

TABLE III

Length of time after beginning of disease that first culture was sent.

1 day	94
2 "	70
3 "	33
4 "	23

5 days	11
6 "	4
7 "	18
8 "	2
9 "	3
10 "	33

Although the vast majority of the specimens for diagnosis are submitted during the first two days after the beginning of the disease, and although it is also true that in many instances the physician has not been called in time to take a specimen during the first few days, this table suggests that in some instances at least there was considerable delay in the sending of the specimens. In diphtheria above all other diseases, where so much depends upon the early administration of anti-toxin and the early isolation of the patient, every means possible should be taken advantage of to make the diagnosis as early as possible.

TABLE IV

Source of Infection

Not known	78
School	36
Family	4
Physician	1

The fact that in the majority of the cases of diphtheria the source of infection is not known suggests that probably many of our cases of diphtheria are transmitted by "bacillus carriers" (see above).

TABLE V

Number of Other Cases of Diphtheria in the House

0	121
1	28
2	17
3	2
4	1

This table speaks well, I believe, for the care used by physicians in limiting the disease to but one member in a family or house in such a large proportion of cases.

TABLE VI

Local Changes

A	Membrane (fibrinous exudate) present	
	Nares	25
	Pharynx	90
	Tonsils	120
	Larynx	16
B	Exudate (sero-mucous) present	
	Nares	22
	Pharynx	63
	Tonsils	114
	Larynx	21
C	No membrane, no exudate, but hyperemia	70
F	No inflammation of any kind	35

This table well serves to illustrate the fact that by no means are all cases of diphtheria characterized by the formation of a distinct membrane. It is true that the figures here given are based upon observations made early in the disease when the specimen was taken for bacteriological examination. It may be that in some cases distinct membranes develop later. It is just as important, however, to remember that all cases of diphtheria are not accompanied by the formation of a false membrane any more than that all pseudo-membraneous inflammations are due to diphtheria bacilli.

TABLE VII

Temperature

98		23	102		47
99		67	103		20
100		44	104		10
101		52	105		1

TABLE VIII

Constitutional Symptoms

Poor appetite	16
Sleeplessness	43
Cough	54
Headache	65
Weakness	30
Pain in neck	31

TABLE IX

Antitoxin used at time specimen was sent

Yes	107
No	139

TABLE X

Number of times bacteria were found in pure culture (practically)

Diphtheria bacilli.....	193
Streptococci.....	142
Staphylococci	215
Pneumococci.....	8

This table is based on all specimens submitted for diagnosis for diphtheria bacilli. As a rule the specimens examined are found to contain a number of different bacteria. Occasionally however they are found in practically pure culture. These figures refer of course to the examination of the material grown on blood serum rather than the examination of the membrane itself.

TABLE XI

Release from Quarantine

A. Number of specimens sent to obtain release from quarantine.

1 specimen	600
2 "	312
3 "	168
4 "	23

B. Release from quarantine in number of days after beginning of disease.

1-14th day	231
15-21st "	250
21-28th "	179
29-35th "	61
36th "	37

In explanation of this table it should be said that the time of release from quarantine was computed from the date on which the physician received the report of the last specimen submitted for release from quarantine. It should also be stated that the rules upon which release from quarantine is based are as follows:—

(Rule 18—Sect. 1, 2, 3—Rules and Regulations, Iowa State Board of Health).

“Rule 18, Section 1. Where possible laboratory findings shall be used to determine the period of quarantine for each individual case of diphtheria. Specimens for culture shall be taken by the attending physician from the nose and throat of each suspected patient, and forwarded to the State Board of Health laboratory for diagnosis. Quarantine and isolation shall be established immediately upon the appearance of suspicious symptoms. In districts where it is not possible for the physician to avail himself of the laboratory tests, and the clinical symptoms are those of diphtheria, quarantine shall be established and maintained for the regular period of thirty-five (35) days, provided however, that, if antitoxin was administered within the first twenty-four hours from the discovery of the initial symptoms, the quarantine may be released at the expiration of twenty-eight (28) days provided the patient has made a complete recovery and these facts are certified to by the attending physician.

Sec. 2. Culture for the release of quarantine shall not be taken until after fourteen (14) days from the date of initial symptoms. When release from quarantine is to be determined by laboratory findings, all infected persons on the premises must show two consecutive negative results from cultures taken synchronously from the nose and throat of each. The second and subsequent cultures for release of quarantine shall not be taken until after the expiration of forty-eight (48) hours after the first or previous culture was obtained.

Sec. 3. All culture examinations for the release of quarantine must be made by a bacteriologist of the State Board of Health, and the reports thereof shall be filed with the local Board of Health. Before the release of any person or persons from quarantine on account of diphtheria, the rules of this Board relative to disinfection must be fully complied with.”

It will be noticed (Table XI-A.) that many patients have been released from quarantine on one examination; many of these represent members of families or nurses who have been exposed to diphtheria and who have been released from quarantine upon a single examination taken after a period of incubation of seven days. In some instances, however, the physician has not complied with the rule of obtaining two consecutive negative reports and has allowed the patient to be

released from quarantine on a single examination. It can readily be understood that in rural communities a strict observance of the law relative to the receiving of two consecutive negative reports cannot always be enforced. When the time comes when physicians and the public in general will appreciate the importance of "bacillus carriers" they must consider it as a duty not only to those with whom the patients intimately associate but to the public at large, to permit no one to be released from quarantine so long as they are the bearers of virulent diphtheria bacilli. Experience has shown that we can not always depend upon a single negative report and that at least two consecutive negative reports should be obtained. Local boards of health should insist upon this important part of the rules of the State Board of Health.

According to Table XI-B, 231 out of 758 or 30.4% were released from quarantine by the end of the second week after the beginning of the disease; 481 or 62% were released from quarantine by the end of the third week and 660 or 87% by the end of the 4th week; only 61 or 8% were kept in quarantine until the end of the fifth week and 37 or 4.8% after the fifth week. The large number of cases released from quarantine within two weeks after the beginning of the disease may be accounted for in part by the number of specimens taken from individuals exposed to the disease but released from quarantine as soon as a negative report was received from the laboratory; in part also by the fact that in a number of cases diphtheria bacilli had entirely disappeared from the nose and throat before the end of the second week after the beginning of the disease. All in all we believe that the rules of the Board that no culture for release from quarantine be taken prior to fourteen days from the date of initial symptoms, is a most important one from the standpoint of the public health.

The figures of these tables, are of great significance when we consider that according to the former "time limit" rule, all cases of diphtheria must remain in quarantine for five weeks, whereas the use of the laboratory findings has compelled only 8% of the patients to remain in quarantine for that length of time.

NEW APPARATUS

Designed (or modified) by Henry Albert

1. MICROSCOPE MODIFIED—PROVIDING IT WITH A LONG STAGE: Bacteriological laboratories such as laboratories of state and municipal boards of health receive a large number of specimens to be examined for tubercle and diphtheria bacilli. Much of the time required for making the examination is consumed by spreading, fixing and staining the material to be examined. This is especially true when the ordinary microscopic slide, sufficient for the examination of only one specimen, is employed.

For some time various laboratories have made use of a long plate of glass (Fig. 1) on which 10 or a dozen spreads may be made, all to be fixed and stained at the same time. The plate of glass may be made from ordinary window-panes. It should be three inches wide and from nine to twelve inches long. It is divided into ten or twelve spaces, each about an inch wide (see Fig. 1), each space to be used for one spread. The spaces may be marked off by means of a diamond or a blue pencil. At the time the spreads are being made a number corresponding to the number of the specimen is marked near one end of each space with a blue pencil.



(Fig. 1)

To facilitate the drying of the spread it is well to place each slide on a small water tank which is entirely closed except one small opening at one of the upper corners. The water in the tank is heated by means of a low bunsen burner. After spreading the specimen it is fixed and stained,

PLATE VII

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the stain being allowed to run over the entire part of the slide which contains the spreads. The subsequent steps are carried out as with the ordinary slide.

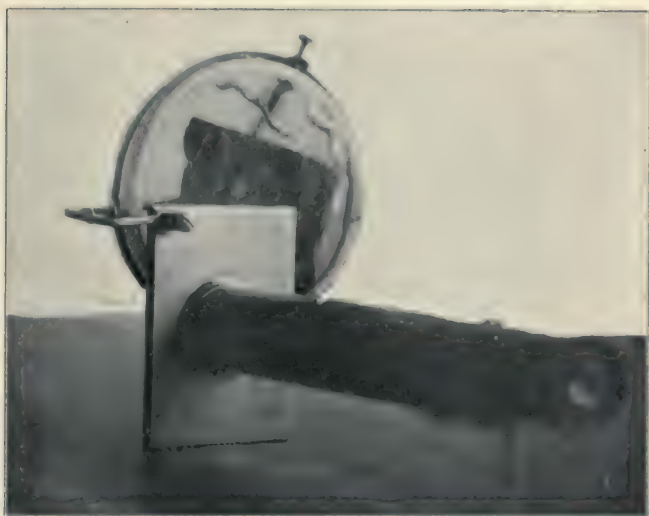
To facilitate the staining of specimens for tubercle bacilli B. R. Rickards, Director of the Boston City Board of Health Bacteriological Laboratory has devised a special staining tank nine inches long, an inch wide and 2 1-2 inches deep with spaces for three of the long slides just described. The slides after having been fixed are placed in this bath, carbol-fuchsin allowed to run in and heated for the required length of time to the steaming point by means of a bunsen burner placed under the bath; the carbol-fuchsin is then permitted to run out and water from tap permitted to run in. By means of a special devise the water will run out of the tank as soon as filled and again automatically refilled when empty. In this way the slide may be thoroughly washed. The slide is then removed and preparation decolorized, washed and stained for a short time with methylene blue, etc., as in the preparation of the ordinary specimen. We have used this tank for two years and have found it to be most satisfactory. After the preparation has once been fixed there is no danger of the bacilli leaving one spread and becoming adherent to another during the staining process. The slides may be used again after having been boiled in a solution containing lye and scrubbed with a cleansing soap or powder. The only objection to the use of a long slide is the difficulty of keeping the slide level on the stage of the ordinary microscope while examining the spreads at the ends of the slide. This we have obviated by having had made, a microscope stage which is twelve inches from side to side instead of three and one-half to four inches as the ordinary stage (see Plate VII). With the use of this stage every spread on the long slide can be examined without any overbalancing due to excessive weight and narrow stage.

The writer is now devising a special mechanical stage to be used in connection with these large slides in order that it may be moved about very slowly and gradually under the field of the microscope.

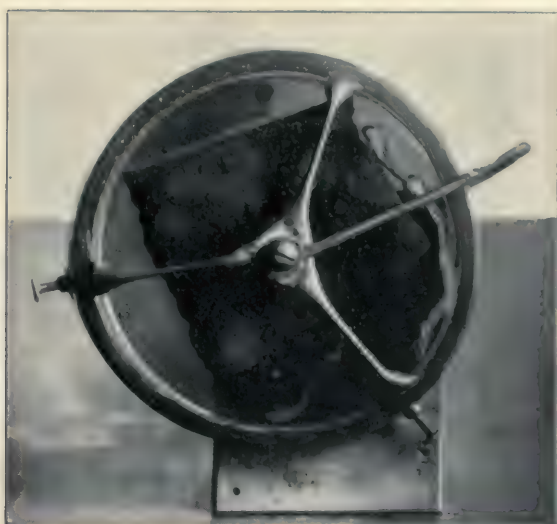
2 DEVICE FOR DEMONSTRATING GROSS AND MICROSCOPICAL SPECIMENS BY MEANS OF THE HAND MICROSCOPE: The writer has improved in some

minor details the device described by him a little more than a year ago. The gross preparation is mounted in glycerin jelly in a Petri-dish made of rather thick glass and the microscopic specimen mounted between two cover glasses is fastened by means of balsam to the base of the Petri-dish in such a way that it is not covered by the gross specimen. I have found that Petri-dishes made of heavy glass and having the base polished are more satisfactory than those ordinarily obtained. The device is designed especially for demonstrating, at one and the same time, gross specimens with microscopical sections, especially on occasions when it is impossible or impractical to use a large stand microscope, as for instance in the clinical amphitheatre, medical societies or in the recitation room. The accompanying illustrations will give a somewhat better idea of the device and the manner of its adjustment. For more detailed description see original article.²

PLATE VIII



View of a hand microscope with the Petri-dish-glycerin-jelly preparation fastened in a special device and so placed as to afford a naked-eye view of the gross preparation. A mounted microscopic section of it is seen at one side.



View of base of hand microscope with a Petri-dish-glycerin-jelly preparation fastened in special device and so placed that the microscopic section is directly under the objective of the microscope.

*REPORT OF A CASE OF BANTI'S DISEASE

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This case was reported by Dr. Bierring and the author at the meeting of the American Medical Association in Boston, spring of 1906. The report on that occasion was with special reference to the blood findings preceding and following the splenectomy. As the patient has died (his death occurring in Nov. 1907) and as we have had an opportunity of examining the remains at post-mortem it might be appropriate to report the case more fully.

The patient, on whose case this study is based, was admitted to the University Hospital, Nov. 15, 1904, and presented the following history:—

C. F. H., aged 38, traveling man, married, having three children in good health, was referred by Dr. W. E. Patterson and entered the hospital complaining of a feeling of heaviness in the abdomen with bloating after meals. Father died at age of 72 years, from hernia; mother aged 72, alive and well; three sisters alive and well; one brother died, aged 44, of typhoid fever. Patient used alcohol, tea, and coffee moderately. He has had gonorrhœa a number of times; denies lues; had common diseases of childhood. He had spinal meningitis at 7 years of age. While in the South, at the age of 21, he had malarial fever, paroxysms occurring, as he claims, only once a week. These symptoms persisted for three of four years, or until patient moved to Colorado, when they soon ceased and have not recurred. There was no history of ague-cake spleen. Patient was jaundiced at the time. In 1890 an abscess developed on the left side of chest, which was opened, and failed to close for

*This paper was received after first forms had gone to press, hence not listed in the Contents.

one year, healing and breaking out in different points on the surrounding area.

History of Present Trouble—In 1902 patient began to feel depressed, had aching and heavy feeling in the left side, with a sharp pain under the ribs on exertion; he continued to work, but nearly fainted a number of times. At Christmas, 1902, his wife first noticed a tumor on the left side of the abdomen. Soon after this the patient consulted Dr. Gilchrist of Iowa City, who made a diagnosis of splenic tumor. In September, 1902, the patient went to Chicago and received several X-ray treatments, but without help, and developed a painful burn on left side of the abdomen over the splenic area, which did not heal for a long time. During the last two years of his life the patient did no work and, except for the burn, felt fairly well. On exertion he tired easily and suffered with dyspnea. There was no hematemesis, but he had frequent attacks of nosebleed; these however had been present more or less since youth. In April, 1904, he had a marked distension of the abdomen for 3 days, for which paracentesis abdominis was advised but not carried out. When examined Nov. 15, 1904, patient was moderately emaciated, had some cachexia, skin, slightly yellow; sclera, jaundiced; pulse 92, regular, fair tension; vessels soft and elastic; systolic pressure 110; heart's area not increased; soft systolic murmur heard over the cardiac area; otherwise sounds were normal. Lungs, normal.

The abdomen was distended, especially in left upper quadrant. There was a diffuse scar on left side, the center of which was unhealed; palpation revealed a large tumor on left side of abdomen which corresponded to the spleen, the lower border of which extended 10.5 cm. below level of umbilicus and the right border 8.5 cm. beyond the midline. The surface was smooth except the right border, which was deeply notched. The liver was distinctly palpable, surface irregular. Small amount of free fluid in the abdomen. Kidneys, not palpable; urine, negative. There were no enlarged lymph nodes. Blood: hemoglobin, 65 per cent. (v. Fleischl); red cells 2,798,800; whites, 3,689. Differential count of leucocytes: polymorphonuclear neutrophiles, 75.5 per cent; small mononuclears, 16 per cent; large mononuclears, 3.3 per cent; eosinophiles, 2.63 per cent; transitionals, 1.12 per cent; mast cells, .37 per

cent; indeterminates, .93 per cent. Daily examinations of the blood were made for 12 days without disclosing any marked change.

Diagnosis—The splenomegaly, leucopenia and secondary anemia permitted a diagnosis of splenic anemia, but the mild icterus, suspicion of ascites and hepatic cirrhosis, which was subsequently corroborated at the operation for splenectomy, made a diagnosis of so-called Banti's disease seem more applicable.

Operation—On November 28, 1904, a splenectomy was performed by Dr. William Jepson. At the operation the liver was found to be enlarged, surface granular and marked by deep irregular scars. About 500 c. c. of clear serous fluid was removed from the abdominal cavity. Recovery from the operation was uneventful.

He was seen by Dr. Bierring in March 1907.⁷ At that time he appeared very well, but there was evidence of a small amount of fluid in the abdominal cavity; there was, however, no discomfort. In the summer of 1907 he began to be troubled with ascites, which became progressively more marked. In July, 1907, it became necessary to aspirate for the first time and from that time on to the time of his death, he was tapped fourteen times. He gradually became weaker and died on November 17 following. The cause of his death was probably asthenia brought on by the recurring ascites.

An autopsy was held by Dr. Albert, who removed the liver, right kidney, lower part of esophagus, the stomach and intestines. The liver was a typical botyroid liver, scarred and nodular, and weighed 1030 grams. The kidney showed a small amount of connective tissue proliferation, however nothing more than might be expected at his age. There was evidence of a considerable amount of hypostatic congestion. The veins in the lower part of the esophagus were markedly dilated, due undoubtedly to interference with the normal flow of blood.

Sections from the liver showed considerable amount of fibrous connective tissue arranged in bands throughout. The liver lobules had lost their normal configuration, the cells being arranged in irregular bands and groups. When stained by Van Gieson's connective tissue method a fine net work of connective tissue appeared to run for the most part along the portal sys-

tem. The cirrhosis might be explained both by syphilitic infection and by being a cirrhosis of the Lænnec type. No distinct gummata could be found. Microscopically the appearance might be explained entirely as Lænnec's cirrhosis, but the gross pathology, being of a typical botyroid type, was so different from an atrophic cirrhosis that I believe a considerable amount of the fibrous connective tissue was due to a syphilitic process. No giant cells were found.

The spleen when removed weighed almost eleven pounds. It was perfectly smooth with a few white areas at the point of contact with the abdominal wall. There were no nodules, scars, or any other evidence present to indicate a syphilitic process, aside from the general fibrosis of the organ.

Sections from the spleen were stained with hematoxylin and eosin; and by Van Gieson's method. All the sections showed a considerable decrease in the number of Malpighian bodies. Where present these bodies were considerably diminished in size and showed some degree of hyaline degeneration. Throughout the whole section could be seen a marked increase in connective tissue. This could be seen growing around the periphery and into the bodies. In the connective tissue meshwork could be seen a considerable number of splenic cells. Open spaces (Hohlraume) lined by endothelial cells, and filled with blood were present throughout.

In Nager & Barmlin's case there was a high grade of hyperplasia of the splenic stroma. There was also extensive sclerosis and circumvascular formation of connective tissue in the blood vessels. Giant cells and eosinophiles were also present.

The anatomical findings in this case corresponds to that described in the majority of cases of so-called Banti's disease. According to Osler, the pathology of the spleen in Banti's disease may represent:—(1) a fibrosis and hyperplasia with atrophy of the pulp and a hyaline degeneration of the Malpighian bodies or—(2) a formation in which the normal texture is largely replaced by fibrous tissue and large endothelial cells with clear protoplasm containing two or more nuclei, and among them giant cells. The latter group forms the so-called Gaucher type of chronic endothelioma of the spleen. The case under consideration comes under the first group.

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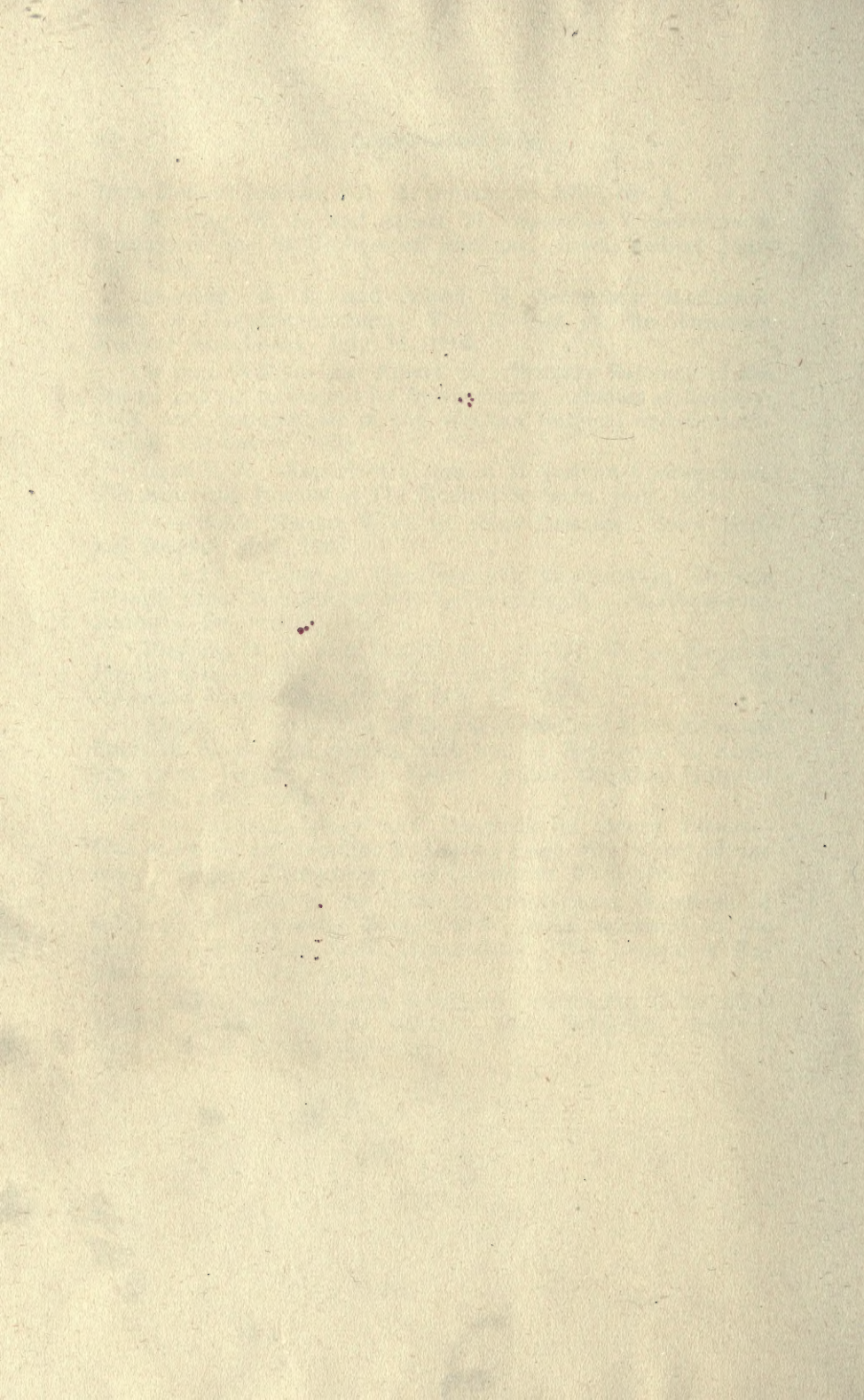
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